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MEDICAL EVIDENCE, CERTIFICATES, AND FEES UNDER THE WORKERS' COMPENSATION ACT.[1]

By R. M. MACKAY, M.D. (Edinburgh),
Chief Medical Referee and Medical Assessor, Workers' Compensation Commission of New South Wales.

A MEDICAL witness differs materially from an ordinary lay witness. He or she has had a university education and has spent many years laboriously studying the human body—its structure in health and disease, and the normal and abnormal functions of its various organs. He has acquired a knowledge of the treatment of the various maladies and accidents from his teachers and has amplified this knowledge by experience. A medical witness gives evidence according to the medical facts (signs and symptoms) that he has observed or ascertained, his interpretation of those facts (diagnosis), and the effect of his efforts to enable the worker to resume his usual occupation (treatment). He is also usually called on to express an opinion as to the "end result" of the accident or sickness (prognosis).

[1] Read at a meeting of the Central Northern Medical Association, Newcastle, on July 21, 1933.

The ordinary lay witness can testify only to what he himself has seen, heard, or experienced, and must on no account attempt to interpret those facts or to draw a deduction from them. That is the function of the judge, jury, or whatever tribunal is hearing the case. I am not for the moment speaking of the expert medical witness. Such may be called on to express expert opinion, not only on the particular patient before the court, but from his knowledge as an expert in similar cases of disease or accident.

It may cause some concern to medical witnesses to learn that in Phipson's "Law of Evidence" (the "Osler" on evidence of the lawyer), at page 386, it is stated:

Value of Expert Evidence: The testimony of experts is usually considered to be of slight value, since they cannot be indicted for perjury, are proverbially, though perhaps unwittingly, biased in favour of the side which calls them, as well as over-ready to regard harmless facts as confirmation of preconceived theories; moreover, support or opposition to given hypotheses can generally be multiplied at will . . . Indeed where the jury accept the mere untested opinion of experts in preference to direct and positive testimony as to facts a new trial may be granted.

Censure of this kind illustrates the necessity for careful consideration by medical men before expressing opinion, and for minute attention to the proved facts on which they are asked to express opinion. The reputation which Phipson accords us is one we shall have to live down.

On a certain abstruse medical question the Judge of the Workers' Compensation Commission recently remarked that eminent medical consultants had expressed one view that was flatly contradicted by other equally eminent consultants. This frequently happens. In a still more recent case there was no unanimity in the opinions expressed in court by eight Macquarie Street specialists as to the cause of a worker's incapacitating condition. Here there was no lack of knowledge, such as the street orator complained of when he shouted: "It's knowledge we want! Ask the av'ridge man when Magna Charta was king of England, and 'e can't tell yer."

Adverting to medical opinion, it may be consoling to remember that His Honour Judge Perdriau stated, in reading a paper before the Medical Sciences Club at the British Medical Association House, Sydney, entitled "Medical Evidence":[1]

> Most text book writers suggest that the expert inclines to the opinion which supports the party calling him as a witness, and, to some extent, his opinion should be accepted with caution. Nevertheless, my experience has been that the majority of those appearing in the Workers' Compensation jurisdiction always endeavour to be fair. This is shown by the fact that very few are engaged exclusively by one side or the other. The expert called for the applicant in one case may give evidence for the respondent in the following case.
>
> By furnishing the tribunal with the best information according to the state of medical knowledge concerning questions in dispute, the members of your profession assist the tribunal in arriving at reasonable and impartial decisions, and thus do justice between the parties. So long as eminent consultants continue to regard it as a public duty to assist tribunals in this manner, the community can have no just cause for complaint in regard to your profession's attitude towards, and interest in, its compensation and industrial problems.

The general medical practitioner, when giving evidence, may be intentionally led by counsel into the realm of the expert, or he may be boldly announced as such after he has given evidence based on his personal experience regarding the case before the court. This is one of the pitfalls, to which I shall refer later.

> On one occasion a general practitioner went into the box with a copy of Osler's "Principles and Practice of Medicine" under his arm. After the usual preliminaries, he was suavely asked by counsel, under cross-examination, if he were a "heart expert". The practitioner replied: "No, but I have had a good deal of experience in hearts." Counsel then read extracts from a number of books, and asked the witness whether he agreed with them or not, finally giving the names of the authors from whom he had quoted. By this time the "heart expert" was obviously "hot and bothered", but was given no mercy by relentless counsel.

Another difference between the lay and medical witness is that the latter is usually privileged to sit in court and hear all the evidence if he so desires. This is a courtesy of the presiding judge, who has power to order all witnesses out of court. It is assumed that medical practitioners are expert witnesses and men of honour, and that anything they may hear in court will be used only to help the court from the medical aspect. This privilege should not be abused.

> In one case before the Commission counsel asked that all medical witnesses should be treated as ordinary witnesses and remain outside the court and the hearing of the court until called on to give evidence. The result was rather startling. There was no unanimity as to the clinical signs on examination, and several diagnoses as to the condition present.

Section 21 of the *Royal Commission Act*, 1923, applies to proceedings before the Workers' Compensation Commission. It provides that: "Any witness before a Commission who knowingly gives false testimony touching any matter material in the inquiry being made by the Commission shall be guilty of an indictable offence and shall be liable to be imprisoned for a period not exceeding five years."

A medical witness should give his evidence slowly and distinctly. Every word he utters is taken down, and if he keeps one eye on the judge taking the notes he will not over-run the rate of writing. This often saves much repetition and may prevent something being recorded which the witness did not mean, or not sufficient attention being paid to important evidence.

The Workers' Compensation Commission is rather unusual in having a presiding judge who is an expert shorthand writer, but medical witnesses would do well to remember that there is often also a medical assessor who is anxious to get down as complete medical notes as possible. Even the court reporter appreciates some deliberation in those giving technical evidence, involving the use of what are termed by the laity "jaw-breakers" and entailing the use of shorthand outlines not in common use. It should also be remembered that the Commission's reporter is not relieved at intervals, as are *Hansard* and Press reporters.

With these preliminary remarks I shall now proceed with the consideration of medical evidence.

In the first place a medical witness should give his evidence without any bias. His duty is to assist the court to ascertain the truth, rather than to lean to any one particular side. As stated before, he comes into court to tell what he found on examination of the patient, his interpretation of the findings, the progress and result of his treatment, and also his opinion as to the ultimate result. He should banish from his mind any preconceived idea that the litigant for whom he is appearing has been harshly treated, that he has taken unfair advantage or that someone is taking unfair advantage of him in some way, or that he is a deserving case or otherwise. The medical witness's duty is to give medical evidence, as defined above, to the best of his ability. Counsel in the case will look after all the other aspects. If the medical witness is a specialist, he should not give evidence outside that specialty, unless he has had special experience in the branch of medicine concerned.

[1] THE MEDICAL JOURNAL OF AUSTRALIA, February 14, 1931, page 187.

One medical witness stated that his evidence on an X ray film (taken by himself) was just as sound as the opinion given by an expert radiologist. While many of us may have a good working knowledge in reading X ray films, it requires considerable self-confidence to assert in open court that we can interpret them as well as an expert radiologist. A medical witness may be frank and say: "I am a general practitioner", and give evidence on medical, surgical, ophthalmic and other cases to the best of his knowledge; but when a witness gives expert evidence in more than one of the specialized branches of medicine it gives food for thought. An expert may give his opinion upon facts which are either admitted, or proved by himself, or by other witnesses in his hearing at the hearing, or are matters of common knowledge, as well as upon hypotheses based thereon.

A doctor may refer to medical treatises to refresh his memory or to correct or confirm his opinion.

Secondly, where possible, a medical witness should bring his original notes to court. In the absence of these he should be perfectly frank when he cannot remember anything. Even when the medical witness has notes, counsel may elect to examine him without reference to them until "his memory is exhausted". The witness may then ask for or be allowed to "refresh" his memory from his notes. A word of warning about these same notes. They must be written at the time the observations recorded are made, and the originals must be used. Notes made "next day" or from memory are notoriously unreliable and usually inadmissible, counsel being not slow to comment adversely on them. No attempt should be made to withhold, alter, or depart in any way from any notes being used. Counsel has the right to ask for the notes, and the discovery of written alteration in dates *et cetera* or verbal alterations of any kind is at once disclosed, often to the medical witness's discredit. I can imagine you all saying that no medical practitioner would do such a thing, but in my limited experience I have seen this occur—and more than once.

In a recent case a medical witness was allowed to refer to an "extract" from his medical records. Very soon he was in difficulties with regard to his dates, but became more positive on his evidence as to facts. When his accuracy on these matters was challenged he became still more positive, and further questioning elicited that he had obtained much of his information from the patient that morning.

Now I think this witness was perfectly honest in his intentions, but such a course was wrong. He was on oath, and should have testified only to matters within his own knowledge or personal experience at the relevant time.

In one case a medical practitioner gave quite lengthy medical evidence, by permission, from the patient's "card". At the end of his evidence counsel asked for the card and after a glance put it in as evidence. The card contained one line, part of which was in reference to the doctor's fee.

When a medical witness is refreshing his memory from a copy of his notes or extracts therefrom, it should be only with the consent of the parties. Opposing counsel may ask if they are full notes, and if not may infer that some important matter has been excluded from the extract. Those medical men who use a card system should bring the original card. Some doctors make their notes in a diary or ledger, and it is quite understandable that they do not desire to carry such a volume into court. However, I draw attention to the importance of using the original notes in preference to a copy of, or extracts from, the original.

Any notes made may be concise, but should be accurate. They should contain the date and history of the accident as described by the worker, as far as possible in his own words, and not as interpreted by the examiner. The side of the body involved should be noted, and if the injury is due to lifting a weight or falling from a height, some attempt should be made to ascertain the nature of the weight lifted or the height fallen. These may seem elementary principles, but it is surprising how often they are omitted and what importance they may assume in a case. It is also advisable to ask what a worker did after an accident, how he got home *et cetera*. I mention this because medical evidence as to the result of being struck by a weight of, say, two pounds falling ten feet would be much discounted if the lay evidence proved that it was ten pounds falling thirty feet. For an interesting example I would refer you to the case of Banecevich *versus* Zinc Corporation Limited [1928], Workers' Compensation Reports, pages 141 to 154.

The worker's description of the accident was that he was pushing trucks of ore along rails. The trucks stood about five feet high. He bent down and on looking up towards a truck about two handfuls of dust from the ore fell a distance of about one foot on to his face and into his eyes. He was twenty-eight days in hospital for treatment to his right eye and then attended for three weeks as an out-patient. He was then told he could go back to work, but he did not resume. Some months later he consulted an ophthalmic surgeon in another State, and the latter, in giving evidence at the first hearing before the Commission, said he had been told that there had been a fall of earth in the mine and a considerable quantity fell on the worker's head and eyes and that his trouble had started then. At a later hearing of the case before the Commission, some fourteen months later, the same ophthalmic surgeon then stated that he had changed the opinion he had given at the previous hearing. He had previously been of opinion that there had been a blow of considerable force from the material which had fallen on the worker in the mine—he thought the material fell from the height of the roof, about twelve feet.

There are some other very interesting features in this case; but what I want to emphasize is the difference between the effects of fine dust falling one foot and mine-roof material falling twelve feet. It has been quite a common experience to find that a medical man has based his opinion as to the severity of an injury on assertions quite opposed to the facts as proved later in a case. If a medical opinion is in relation to particular machines or occupations a medical witness should give such opinion only when he has a personal working knowledge of the machine or occupation.

In a recent case a medical witness gave evidence that the applicant's condition could have arisen as

described, namely, a movement on a machine repeated hundreds of times daily over a period of many weeks. When it was shown in evidence that this continuous use of the machine was not a fact and that the machine was used only for short periods at intervals, the medical practitioner's opinion—based on wrong premises—was valueless.

Thirdly, a medical witness must keep his temper—and this is sometimes very difficult. Counsel is quick in discovering a "touchy medico" and soon a question is asked which arouses indignation, and then the unhappy witness usually says things he would not dream of saying had he kept cool. It is wise to say as little as necessary and answer with a direct "yes" or "no" wherever possible. Examining counsel does not want explanations which do not suit his case, but if counsel calling the doctor is alert he will see that the doctor is not placed in a false position by such answers and will give him an opportunity to express himself later in reexamination.

Fourthly, a medical practitioner who adopts an indifferent or supercilious manner in the witness box is looking for trouble. A certain decorum is observed in all British courts, and the lounging, casual witness makes a bad impression on the bench, whilst the flippant medical witness is soon in trouble with the bar. If a question asked by counsel is involved or not quite clear, a medical witness is quite in order if he asks that the question be made clear. Some counsel have a habit of asking several questions in one, and a witness may wish to say "yes" to part of the question but would say "no" to another part if they were asked apart.

Fifthly, avoid recriminations in the box, however much you may be tempted to indulge in them. They are in bad taste and unconvincing.

In one case I was asked to examine a litigant with his own medical practitioner during an adjournment. We agreed on every point, even to prognosis. Imagine my surprise when, on returning to the box, my colleague gave evidence totally different from what we had agreed on in consultation. My first impulse was to voice my disapproval, but on second thoughts I just stated what I had found clinically at the consultation.

In a case before the Commission two medical witnesses gave evidence which was diametrically opposed. When the second witness had concluded, the disparity in their evidence was pointed out to him by counsel. He then blazed forth: "Yes, I know the reason. Dr. X and I were in opposition when we both practised at Z, and when I left . . . ." The story was interrupted by counsel saying: "Thank you, Doctor, that will do."

Many of you have had much experience and are well qualified to take care of yourselves in the witness box. To the younger members I would tender a word of advice. We are none of us infallible, not even the youngest of us. Never be cocksure of yourself when going into the witness box. One of the ablest medical witnesses I know has confessed that even now, after many years' experience, he is still very nervous at first when giving evidence. Only when properly "set" does he feel at home under cross-examination. A case comes to my mind which may illustrate what I mean.

A young doctor, who had been announced as being on the staff of a leading hospital, had given evidence on a rather involved medical matter. Under cross-examination counsel appeared to be so kindly and benevolent that the witness was led into "spreading himself" and airing his views, which were contrary to expert opinion, somewhat expansively. This continued for some time, and the examinee had sailed serenely into deep waters with concealed reefs all round. Counsel then, apparently as an afterthought, asked: "Doctor, you are on the staff of . . . . Hospital?" "Yes." "What is your position?" "House surgeon." "How long have you been in that position?" "Three months." "How long have you been qualified?" "Six months." "Thank you, Doctor." The ensuing momentary silence in the court spoke volumes.

In the recently published work, "Lord Darling and His Famous Trials", reference is made to "The King *versus* Armstrong", when a solicitor was convicted of wife poisoning by arsenic.

Mr. Justice Darling's charge to the jury on that occasion was considered such a masterpiece that it is set out *in extenso*. Dealing with the evidence of the medical experts he remarks on pages 235 and 236:

Now we come to the contest between Dr. Spilsbury and Sir William Willcox on the one hand, and Dr. Toogood and Dr. Ainslie and Dr. Speed on the other. First of all, look at the evidence of Dr. Spilsbury, because he really saw more of this case than any other doctor except Dr. Hincks. Dr. Spilsbury never saw her alive; he was called in for the post-mortem, and Dr. Ainslie was present, representing the defendant, and saw what was done, so he saw then the condition of things just as Dr. Spilsbury did, although Dr. Spilsbury conducted the post-mortem . . . . I do not think I need go into this minutely because the defence agree that she died of arsenical poisoning . . . .

Let us consider who these doctors are. It is for you, you have been told you are the judges of this case, not I. Do you remember Dr. Spilsbury, do you remember how he stood and the way in which he gave evidence? Do you remember or do you not remember how, if there were any qualifications to be made which told in favour of the defence, he always gave it without being asked for it? Did you ever see a witness who more thoroughly satisfied you that he was absolutely impartial, absolutely fair, absolutely indifferent as to whether his evidence told for the one side or the other, when he was giving evidence-in-chief or when he was being cross-examined? You should recollect and consider the demeanour of every witness in every case that you try; it is most important . . . . As to what he said, you will judge whether you agree with it, whether you think it well founded or do not.

Medical Certificates.

Webster defines the word certificate as "a certified statement; a written testimony to the truth of any fact". Bearing in mind this definition—a written testimony to the truth of any fact—we must guard against ambiguity or inaccuracy in writing certificates. I would also remind you that it is a well recognized legal principle that the *suppressio veri* is just as bad as the *suggestio falsi*, that is, concealment of a material fact is equivalent to intentional misrepresentation, and either is a fraud on the party relying upon the veracity of the other party.

In respect to medical certificates I must repeat what I have considered to be essential in medical notes—they should be concise, correctly dated, and accurate. Many doctors give weekly certificates, each stating that the worker will be unfit for work for one week. These are probably intended for lodge purposes, but are frequently used in court, and unless the series is complete may result in injustice

to a worker. It seems to me, but here I am open to correction, that it would be preferable to say "is in my opinion unfit to follow his usual occupation". The incapacitating condition should, of course, be stated. I am afraid that medical certificates are often issued without sufficient care by some medical practitioners. Of course, we know that many patients consider they are not being properly treated unless they get "a bottle and a 'stifcate'". It is also very unwise to issue duplicate certificates unless they are worded identically with the original and marked "duplicate".

I must now refer to a much more serious matter, and that is the issuing of postdated or antedated medical certificates, and to corrections made in originals at a subsequent date. Unfortunately I have seen examples of all these practices, which cannot be too strongly condemned. Such things may have been done in good faith, but when under review months or years later can be viewed only with suspicion. In short, never put in a medical certificate what you are not prepared to support on oath. If the certificate turns up years later and you forget all about it, be candid and say so. When issuing a medical certificate it is well to remember that it is a document that may be produced in court months or years later, and its contents may have far-reaching effects. For example, if you include in the certificate the nature and date of the accident, preface it with: "the patient states that" or "the condition found is consistent with the history of" *et cetera.*

It is a routine practice with business firms to keep a carbon copy of all outward correspondence. Would it not be possible for doctors to use interleaved books for their medical certificates and, by means of carbon paper, to keep a facsimile of their certificates? I would also suggest that rather than antedate a certificate, head it with the date on which it is written and state that the worker may resume on a date given. The practice of antedating certificates has probably arisen from the frequency with which medical certificates are given on a Friday, to enable the worker to resume work on the following Monday. If this be the reason, the certificate should bear Friday's date and the date be stated on which the worker may resume work. To prevent any ambiguity as to the date of resumption, the certificate should read: "may resume work on . . . (date)". If the certificate states will be "fit" on, say, the 23rd, workers frequently start work on the 24th.

A medical certificate should never be given bearing a date unless the practitioner has seen the patient on that date. Quite recently a case was brought under my notice in which an inland country practitioner forwarded weekly certificates regarding a patient receiving compensation for an injury. The said patient was at the seaside, some hundred miles distant, while these medical certificates were being furnished. Now I find it difficult to justify such a course. The practitioner said in explanation that he knew it would take the injured worker six weeks to recover sufficiently to resume work; but I do not think that justifies giving medical certificates *in absentia.*

On one occasion, many years ago, I received a medical certificate from a recently qualified "M.B., Ch.B." stating that the worker was "suffering from strained muscles of the right shoulder and back through having been struck and knocked over by a bag of flour coming down a chute on (a date given)". The case had been listed for hearing in the District Court, and as I had definite evidence that the injury was due to an entirely different cause, I saw the medical practitioner who had given the certificate. At first he was inclined to stand on his newly acquired dignity (we have no doubt all passed through this stage after graduating). He said he was quite prepared to support his certificate on oath in the witness box, although it would be his first appearance there. I then asked him if he had been present and seen the accident. He replied that he had not, and when I pointed out that he had certified to what were questions of fact and not medical matters and could be called on to substantiate them, he implored me to give him back the certificate. After a very friendly discussion on medical certificates we parted the best of friends, and I think he will not give a loosely worded medical certificate again.

Quite frequently medical certificates are given which state that certain voluntary movements by the patient or palpation by the writer of the certificate cause the worker pain. Now pain is an abstract thing, except to the person feeling it, and no medical practitioner can feel the pain he may elicit during an examination. In a medical certificate always say, not "causes pain", but "causes a complaint of pain". The same rule applies in giving evidence. Such advice may appear pedantic, but carelessly worded certificates are apt to convey the impression of inexactitude in other respects.

If you have given a medical certificate through being misinformed, or in error, candour is better than trying to "cover up your tracks".

On one occasion, after a doctor had given evidence regarding a certain injury, based on his reading of an X ray film, counsel triumphantly confronted him with his own certificate giving a quite different diagnosis. He at once admitted that the certificate was his and that he had made a mistake in his original interpretation of the X ray picture. Some time after giving the certificate he had submitted the X ray film to a radiologist friend who had pointed out his error. In the meantime he had lost touch with his patient, and until confronted with the certificate in court had had no opportunity of rectifying the matter. Now it requires some pluck to acknowledge publicly a mistake. I was much impressed, and have no doubt the members of the Commission were also, with the integrity of this witness.

Medical Fees.

We now come to the most debatable division of my paper, namely, that dealing with medical fees in workers' compensation cases. First, let me take you back to the conditions existing before the inclusion of medical benefits in the *Workers' Compensation Act* of 1926 and its amendments. Prior to this Act an injured worker who was incapacitated for more than one week was paid compensation at a maximum rate of £3 per week, with a maximum total of £750. The employer was under no further obligation and the worker sought medical advice and treatment where he could best obtain it. Thrifty workers joined lodges or medical benefit

societies, but many simply went to the nearest public hospital, and to the credit of these hospitals treatment was freely given. This good work was so generally recognized that many employers voluntarily contributed financial support to the hospitals and many workers also assisted by special workshop collections, hospital fêtes, hospital Saturday, and in other ways. Treatment in hospital of this class of patient was accepted as a matter of course by the honorary staffs, the members of which, I am sure, made many good friends among such workers. Subsequent to the medical provisions of the *Workers' Compensation Act* of 1926 this was all changed. The maximum weekly rate of compensation was raised to £5 per week and the total liability to £1,000. The administration of the Act was vested, as you are aware, in a Commission consisting of a judge and two members, compulsory insurance was introduced and incapacity resulting from injuries, whether by accident or not, but not intentionally self-inflicted, became compensatable so long as there was a causal connexion with the employment. ("Injury" was defined to include diseases other than those caused by silica.)

It would take too long to discuss in detail all the changes brought about, and I will confine myself to those chiefly affecting our profession. In addition to weekly monetary compensation payable by the employer to an injured worker during incapacity, the employer now has to provide, when necessary, medical or hospital treatment or ambulance service under Section 10 of the 1926-1929 Act.

For the purposes of this section:

"Ambulance service" includes any conveyance of an injured worker to a medical practitioner or to a hospital.

"Medical treatment" includes:

(*a*) treatment by a legally qualified medical practitioner, a registered dentist, or a masseur; and

(*b*) the provision of skiagrams, crutches, and artificial members; and

(*c*) any nursing, medicines, medical or surgical supplies or curative apparatus, supplied or provided for him otherwise than as a patient at a hospital.

"Hospital treatment" means treatment at any hospital and includes the maintenance of the worker as a patient at the hospital, and the provision or supply by the hospital of nursing, attendance, medicines, medical or surgical supplies or other curative apparatus, and any other ancillary service.

The maximum sum for which an employer is liable for hospital or medical treatment was fixed by the 1926-1929 Act at £25 in each case. In addition, the cost of hospital treatment was fixed at £3 3s. per week for in-patients and for out-patients at 3s. per treatment, with a maximum of £1 1s. per week.

The amounts payable for the loss of certain members of the body are specifically set out in Section 16 of the Act, and provision was made for examination of injured workers by medical boards and medical referees appointed by the Commission. Under its powers the Commission can summon a medical referee to sit with it as medical assessor, and a practice has been made of doing so in all cases involving medical evidence. This, I think, must be regarded as very desirable, and has raised the standard of medical evidence given before the Commission. But I am digressing.

The Workers' Compensation Act of 1926 provided that the cost to the employer of medical, surgical and hospital treatment of his injured worker be limited to £50 unless the Commission otherwise directed. Having regard to the particular facts of each case brought before it, the sums allowed by the Commission varied from approximately 10s. 6d. to £150. The Act was amended by the Legislature in 1927, and public hospitals were given the right to claim the cost of hospital treatment from the worker's employer; but the employer's liability for medical, surgical and hospital treatment remained as above.

The first and outstanding effect of the new legislation in 1926 was to remove a very large proportion of our working population from the status of what I may term "hospital patients" to the "paying class". As the public hospitals were for the relief and treatment of "indigent patients", the new Act had far-reaching effects. Hospitals did not admit potential paying patients as indigents and, in practice, after giving such immediate treatment as was necessary to injured workers, referred them to their own medical advisers. The members of the honorary staffs considered that they could not be expected to treat potential paying patients as indigents, and what medical practitioner with any business acumen would allow a patient potentially worth £50 past him? I do not wish to be unkind, but was it more than coincidence that so many of the early medical accounts under the provisions of the new Act approximated to this sum?

While in the majority of cases the cost of this treatment was covered by insurance and paid to the doctors and hospitals by insurers, the large sums which insurers were called upon to pay were reflected in the insurance premiums charged to industry, and no doubt industry complained of the extent of the burden it was being called upon to bear. As a result, in 1929, the medical and hospital provisions of the Act were again amended, and this time the benefits were reduced. The Commission was no longer vested with the right to award medical, surgical or hospital costs in very serious cases beyond the prescribed aggregate limit; and this limit of £50 was divided into a maximum of £25 for medical treatment and £25 for hospital treatment. Costs incurred beyond these sums have to be borne by the injured worker. Since the passing of that amending Act in 1929 industry has suffered a very serious financial set-back owing to the world-wide depression, and it has been rumoured in some circles that the costs which industry could bear in 1929 are very different from those which it can bear today. This is an aspect which might, if medical costs are heavy, weigh with the legislature if and when consideration is again given to amending the Act. The fable of the goose and the golden eggs is very much in point.

We now come to a most important clause, namely, Section 10, Subsection 4, Clause (*a*):

The sum for which an employer shall be liable in respect of the medical treatment of a worker shall be such sum as is reasonably appropriate to the treatment afforded, having regard to the reasonable necessity for such treatment and the customary charge made in the community for such treatment to persons other than workers.

This somewhat involved sentence must be analysed in any attempt to comprehend its meaning. The sum for which an employer shall be liable in respect of the medical treatment of a worker shall be such sum as is reasonably appropriate to the treatment afforded, having regard to: (i) the reasonable necessity for such treatment, and (ii) the customary charge made in the community for such treatment to persons other than workers. Now in regard to (i), it cannot be considered a reasonable necessity for a practitioner to visit daily a worker in hospital with a minor injury who is receiving appropriate hospital treatment from nurses or attendants trained to such work and competent to report any change in the condition requiring attention. Nor can "reasonable necessity" be claimed for seeing daily a patient with strained muscles of the back or, say, a sprained ankle, or when a worker is convalescent.

On one occasion a medical practitioner gave evidence before the Commission that, after having diagnosed sprained muscles of the back and prescribed a liniment, the main curative agent was rest. Yet this same practitioner within a few months charged for daily attendances on such a patient up to the day before the worker resumed duty.

Nor can it be considered "reasonable treatment" to keep an injured worker in hospital longer than is warranted by the severity of his injury. Perhaps this is more likely to occur in the country, where the attending practitioner may consider that the distance of the worker's home or possibly his home surroundings are undesirable factors. When this is the case I would suggest that the doctor inform the employer or insurer at once of the reason. If the employer or insurer declines the added expense of the suggested longer hospital treatment for the reason given, and if as a result any untoward effect the medical practitioner feared occurs, then the employer or insurer would probably be called upon to carry the extra burden. An employer or insurer would, I think, appreciate that the medical practitioner was only suggesting something in the best interests of all parties.

In one account referred for conciliation it was suggested that a worker with an injured hand in a short-staffed country hospital had proved himself so useful to the nurses (and incidentally to the doctor) that he was kept in hospital many weeks longer than was necessary. Can this be defined as "reasonable treatment"? There was a certain amount of evidence both ways, and eventually a compromise was effected. But all this controversy would have been avoided had the medical practitioner done as advised above.

We shall now deal with the next clause. You will observe that this clause is also governed by the introductory sentence, namely, that the sum for which an employer shall be liable in respect of the medical treatment of a worker shall be such sum as is reasonably appropriate to the treatment afforded, having regard to the customary charge made in the community for such treatment to persons other than workers.

It is difficult for a layman to define what portion of the community are "persons other than workers", but I suppose one would be safe in assuming that hoboes, millionaires, members of Parliament, clergymen, old age pensioners, and our wives are "persons other than workers", and as each of you is quite familiar with the "customary charge" made to these sections of the community we have a clear basis to start on. For example, was it Pierpont Morgan, Vanderbilt or Henry Ford who was willing to pay $1,000,000 to any doctor who could permanently cure his indigestion?

Some years ago a dental journal quoted the case of an American dental surgeon who charged a Continental princess—who comes into the category of a person other than a worker—the equivalent of at least £100 in dollars for each of two gold stoppings. At that time the fee in Macquarie Street for similar stoppings was one guinea each.

As a matter of hard fact we know that many practitioners make no charges to hoboes and clergymen, and that the charges to wives must bear some relation to the husband's ability to pay them. I will at this stage confess that a "worker" has been defined in the Act, and that "other than workers" probably means a patient not entitled to compensation under the *Workers' Compensation Act*, 1926-1929, so that our primary assumption is not so very far out. "Persons other than workers" include persons whose remuneration exceeds £550 per year, the police force, unemployed, unemployable, the aged; and many others will occur to you. I think it clear that our charges to the employer for the reasonable treatment of an injured worker must have regard to the customary charge to those "persons other than workers". I think it would not be reasonable to charge a seaman the same fees as a solicitor; a laundryman the same as a land owner; a milliner the same as a merchant; or a dustman the same as a director of an insurance company. We must, and in practice do, recognize this, and vary our fees accordingly. I may illustrate this point by quoting from evidence recently before the Commission.

The worker was a labourer in casual work and said in evidence that he owed the doctor £6. The doctor had said to him: "It ought to be £6, call it £2 and I will not press you for that." The doctor in his evidence said that his full charge for treatment of the injury was £6, but he had only charged £2 4s. Regarding the 4s., he had said to the worker: "Buy a lottery ticket with the odd shillings and if you win we will go halves."

How can we recognize fees amounting to, say, £3 3s. per week as being reasonably appropriate to a domestic servant receiving 10s. or 15s. per week? In other words, the employer is only liable to

indemnify the worker for the cost of treatment which he (the worker) would otherwise be called upon to pay, and this is the dominant principle governing medical costs in workers' compensation matters. The purpose of the Act is to compensate a worker for loss of earnings and to indemnify him for medical costs in which his injury involves him. It gives a medical practitioner no right to claim the cost of treatment from the employer and, in fact, vests no rights whatever in medical practitioners to demand payment of fees. Of course, the practitioner has his common law rights against any patient for the recovery of fees. The Act is solely what its title purports it to be—a workers' compensation act. While bestowing on the worker medical and hospital benefits payable by his employer, it also places limits on the employer's liability, one of which is that his liability in respect of the payment to the worker of his medical costs shall be no more than an indemnity, and that indemnity shall not exceed £25, no matter whether the injury is one that may involve intensive treatment over a prolonged period.

I now propose to pass round an X ray picture for your inspection and should like your opinion of what would be a reasonable charge for treatment. The history is as follows:

While stepping from a wharf on to the railing around a lighter the worker took hold of the rigging in his right hand to steady himself, but his foot slipped off the railing. He fell to the deck, landing on his right side. He carried on at work for half an hour, but, as he could not then put his right foot on the ground, was taken to hospital and his condition diagnosed as "severe contusion right thigh". He was then at home three days, and on the fourth day after the accident he saw a doctor. There were contusions and abrasions of the antero-lateral aspect of the lower part of the right thigh and some synovitis of the right knee. An X ray picture was then taken, as the patient was complaining that the leg was very painful on walking. The radiologist's report was: "There is a fracture of the cortex in the upper third of right femur, in good position." After the X ray examination the patient returned home and called in his own doctor. He was unable to work for eight weeks. During the period of incapacity he was visited by his doctor sixteen times. (The doctor's account reads: "Fractured femur, as per Schedule D—£21.")

The following cases may be of interest:

In a recent case heard before the Commission the medical and hospital expenses amounted to £37, while the total compensation received by the worker was £39.

In an account recently submitted to the Conciliation Branch of the Commission, the hospital and medical expenses asked for totalled £51 (the medical attendant asked for £33) and the worker received £22 as compensation.

In a still more recent case the medical expenses were £10 10s. and the total compensation paid to the worker was £6 8s. 6d.

In a further case before the Commission the medical expenses were as follows:

Hospital, 15 weeks	£47
Chemist	£2
Doctor (96 visits)	£49
Total	£98

The worker, who was suffering from a septic leg, was earning 30s. per week. It would have taken him thirty-two and a half weeks to earn enough to pay the doctor, supposing that he handed over all his wages.

Now what chance would a doctor have of recovering such fees if he had to depend for payment on the worker alone? I could quote many similar instances.

In conclusion, I should like to draw your earnest attention to page 73 of the "Sixth Annual Report of the Workers' Compensation Commission". The Government Statistician there points out that the proportion of the cost of medical treatment to compensation payments to the worker has increased each year since the passing of the 1926 Act, and this notwithstanding the amendments to the Act which I have already mentioned. The costs were:

1926-1927 ...	4·09%	1929-1930 ...	9·66%
1927-1928 ...	7·25%	1930-1931 ...	10·46%
1928-1929 ...	8·63%	1931-1932 ...	12·13%

When comparing 1931-1932 medical costs with weekly compensation payments alone, that is, excluding lump sum or death payments, the percentage is higher still, being 13·31%.

It is an indisputable fact that the medical provisions of the *Workers' Compensation Act* have diverted annually a large sum of money in the aggregate to the medical profession. To take the simile of a gold mine, it has converted unproductive over-burden and reef into payable ore—low grade if you will, but payable. And our mining experts are now treating refuse dumps and low grade ores, which in better times they would have scorned.

DIABETES MELLITUS.[1]

By EWEN DOWNIE, M.D. (Melbourne), M.R.C.P. (London),
Physician to Out-Patients, Alfred Hospital; Physician to Austin Hospital, Melbourne.
(From the Baker Institute of Medical Research, Alfred Hospital, Melbourne.)

THE successful isolation of insulin some ten years ago has been responsible for a new and increased interest in the subject of diabetes. Our ideas of treatment, of diagnosis, and of prognosis have undergone a radical change and are still in a state of flux. Our knowledge of other aspects of the disease, however, has not altered so rapidly. Of the nature of the causation of diabetes we are still ignorant. I do not propose to indulge in any long historical survey, but I would remind you that the modern ideas on the subject are based on the work of Claude Bernard, who some seventy years ago showed that there was a close relationship between glycosuria and an excessive sugar content of the blood. Then followed the discovery by Langerhans of certain groups of islet cells in the pancreas that now bear his name. Von Mering and Minkowski soon after produced fatal diabetes in dogs by total extirpation of the pancreas, and in 1895 Schafer suggested the connexion between the islet cells and diabetes. Now that insulin has been isolated and

[1] Read before the Gippsland Subdivision of the Victorian Branch of the British Medical Association on December 18, 1932.

its effect in controlling severe diabetes is known, we are still in ignorance as to why such patients develop a failure of their insulin supply. Much speculation as to islet degeneration and regeneration has been made, but at the present time we do not know why diabetes develops or how it commences. Intensive research, stimulated by the discovery of insulin, has thrown some light on carbohydrate metabolism, but further work is necessary before the complete explanation is known.

Recent work is attracting attention to the part played by heredity. In many cases of diabetes in which other apparently normal relatives have been investigated by blood sugar curves, evidence of a diabetic taint running through the family has been revealed. This has prompted White to state that diabetes is probably transmitted as a Mendelian recessive. From the clinical side the influence of heredity in this disease has been often overlooked, but it is rapidly assuming importance.

Definition.

The exact definition of *diabetes mellitus* is difficult for the reasons just mentioned, but it can be defined as a disease characterized by a disturbance of carbohydrate metabolism, resulting in an increase in the amount of sugar in the blood and in the excretion of sugar in the urine, probably arising from a failure of the pancreas to produce insulin in adequate amounts.

Ætiology.

Diabetes can occur at any age. It has been observed in the earliest months of life and has made its first appearance in rare instances after the age of eighty. Comparatively speaking, it is uncommon up to middle life, and in these earlier age periods it affects the sexes equally. From forty-five onwards it is more common and affects the female sex more frequently in the proportion of two females to one male.

In the majority of cases no predisposing cause can be determined. Occasionally diabetes develops during an acute toxæmia or during pregnancy.

Symptoms.

The onset of diabetes is variable. In 75% of all cases the onset is indefinite and by that I mean that the patient is unable to give a history of onset which can be placed within a period of two months. In 18% the onset is gradual over a period of one to seven weeks, while in the remainder the onset is acute and can be placed within one week. In general, the earlier in life the disease occurs, the more sudden its onset; and in general, the diabetes of early life is more severe than that of middle age. To both of these statements, however, exceptions are to be found.

In the acute case there is usually a typical history of the sudden development of excessive thirst, polyuria and a fairly rapid loss of body weight. Ravenous appetite may or may not occur at this stage. As the disease progresses, marked loss of energy is the rule and the patient is easily tired. Such patients, if untreated, may progress to diabetic coma within a few weeks. Acute diabetes may develop at any age, but is usually seen in young people.

A rather different picture is presented by the mild case usually in middle life. There is often a history of a fairly rapid gain in weight (the so-called preglycosuric obesity) over a period of two years or more. This obesity may remain for several years and then the weight commences to fall, often to the patient's relief, but it continues until the patient becomes alarmed and seeks medical aid. Sometimes loss of weight is not a marked feature, and lassitude and tiredness may be the first symptoms. Again, the condition may be so insidious in onset that attention is first directed to diabetes by the occurrence of one of the complications of the disease, such as cataract or gangrene. *Pruritus vulvæ* is at times the initial symptom and advice may be sought for this alone. Lastly, a certain number of the mild cases are first discovered on routine examination of the urine during life insurance examinations.

Diagnosis.

In any case of suspected diabetes the testing of urine for sugar is undertaken, and it is necessary to consider this question a little more fully in dealing with the subject of diagnosis.

The diagnosis is usually obvious when glycosuria occurs in conjunction with a rapid history of thirst, polyuria and loss of weight. However, in many of the milder cases and in cases of symptomless glycosuria further evidence is necessary. At this stage I would emphasize that, although we know that certain glycosurias are benign and require no treatment, it is quite impossible to determine by the history and urine examination alone which cases are diabetic and which are not. I would further stress the necessity of considering every glycosuria as diabetic until the contrary is proved. The attitude of neglecting a glycosuria because it is slight in amount or because it is not constantly present is to be condemned.

In these cases of doubt help must be sought from determination of the sugar content of the blood. The blood sugar content is never stationary over any period of time, but rises and falls within certain limits following the ingestion of carbohydrate food. In the diabetic, with his inability to deal with carbohydrate food, the blood sugar is usually raised before treatment is instituted. In the doubtful case it is usual to test a patient's ability to deal with carbohydrate in the following way. The patient fasts overnight and in the morning voids urine, which is retained and tested for sugar. A sample of blood is then collected for estimation of its sugar content. The patient then takes 50 grammes of glucose, which may be dissolved in a tumbler of water and flavoured with a little lemon or orange juice, and samples of blood are collected at half-hourly intervals up till two

hours after the taking of the glucose. A second sample of urine is then obtained and tested for sugar. The results of the blood sugar estimations are plotted in the form of a curve. Normally the fasting blood sugar lies between 0·08% and 0·11%; the curve reaches its peak, which should not exceed 0·17%, within half to one hour and falls to the fasting level in one and a half hours. The diabetic usually shows a raised fasting blood sugar; the curve rises to a high level and falls slowly, often not reaching the fasting level for some hours.

Certain cases, although showing normal blood sugar curves, are associated with glycosuria, which is marked in the two-hour specimen of urine. These cases are termed renal glycosuria, because the renal threshold to glucose, which normally lies between 0·17% and 0·18%, is lowered, allowing the appearance of sugar in the urine, although the blood sugar is within normal limits.

Other cases show a curious response to glucose. The blood sugar in the fasting state is normal, but the half-hour sample of blood may show a high reading, sometimes well over 0·20%. The hour sample is, as a rule, back to the fasting level. These cases, which are termed lag-curve glycosurias, are thought to be due to a lag in the production of insulin or to rapid absorption of the glucose. Such cases are usually associated with intermittent glycosuria.

A case of lowered renal threshold or of lag-curve glycosuria is considered at the present stage of our knowledge to be benign and does not require any treatment. It is wise in any case of doubt to subject such patients to a periodical review.

Facilities for the estimation of the blood sugar are not available to many medical practitioners who are situated away from hospital centres and, in consequence, the diagnosis of a symptomless glycosuria may present some difficulty. A rough but reliable method of diagnosis is described to overcome this. The patient fasts overnight. At a stated hour in the morning he voids the urine. He then takes by mouth fifty grammes of glucose dissolved in water. Punctually one and a half hours later he attends his doctor, who collects one sample of blood in a Behring venule containing sodium fluoride. A sample of urine is also obtained and tested for sugar. The venule may be packed and sent by post to the nearest laboratory. The result is interpreted thus: If the blood sugar is below 0·12% the condition is not diabetic, and if sugar is present in the sample of urine obtained the case is one of benign glycosuria. If, however, the blood sugar is above 0·12% the condition is probably diabetic, and if above 0·16% it is certainly diabetic.

Treatment.

Before commencing any detailed consideration of treatment certain general principles may be laid down.

The first essential is that, whatever line of treatment is adopted, the patient should be given a diet which will be adequate for him to maintain his weight and strength, not only while he is in hospital, but while at his ordinary work. If this is not done, the patient is tempted to break diet, often with serious consequences, and he is not always to blame.

Secondly, as diabetes is at the present time an incurable disease, education of the patient in the nature and control of his disease is an essential part of his treatment. In this respect diabetes differs from almost all other diseases, because the more a patient knows of his disease, the better it is for him. A certain number of patients resist treatment, declaring that they cannot be bothered with diet, and state that if they require insulin they would sooner be dead. Such patients present difficulty because of their mental outlook, but if this cannot be overcome they have the satisfaction that sooner or later, despite the efforts of their doctor, their wish is gratified and they die ingloriously in coma. Such cases are fortunately rare.

Thirdly, diabetes is a disease and should be treated as such. The patient with mild diabetes, if told merely to abstain from bread, potatoes and starch, is surely ill advised. Meticulous care and attention to detail are the only ways of obtaining satisfactory results, and even if the patient's intelligence be slight, he should be given proper instruction at the outset, so that he is at least given a chance.

As a preliminary to indicating the application of diet to the control of diabetes, let me first indicate the principles upon which the diabetic diet is based. Three principles may be considered: (i) restriction of the total diet, (ii) restriction of carbohydrate and protein, (iii) the balancing of the total carbohydrate and the fat in such a way as to allow sufficient carbohydrate for the proper metabolism of the fat.

1. Restriction of the total quantity of food is a common sense procedure. The majority of the human race eat more than their daily requirements, and the diet of any diabetic should be so arranged that it is sufficient for him to maintain his weight and allow him to work, but no more.

2. Restriction of the carbohydrate is again a measure of common sense. If a person shows inability to deal with an unlimited amount of carbohydrate, it should be restricted. Restriction of protein down to the figure of 1·0 to 1·5 grammes per kilogram (two and one-fifth pounds) of normal body weight is advisable, because excess protein raises the basal metabolic rate, with a consequent demand for a higher total diet, and secondly, 58% of protein food is capable of conversion into sugar in the body.

3. If an individual shows an inability to deal with carbohydrate foods the question arises, why not omit them entirely and live on protein and fat? Apart from the fact that such a diet would be distasteful to many, there is the grave objection that, if adequate carbohydrate is not allowed, the

fats are incompletely metabolized and a condition of acidosis, or more correctly ketosis, develops.

The next step is to consider the application of these principles to the control of the diabetic patient.

1. If the total diet is to be restricted, how is this to be managed? Without discussing any elaborate calculations, it is known that for each kilogram of body weight an adult requires at least 25 to 30 calories per day, the calorie being an arbitrary unit of energy. With a man engaged in hard physical work the calorie requirements naturally rise up to 40 or more per kilogram per day. The calorie value of any diet is obtained by the following formula, the quantities of the constituents being expressed in grammes:

$$\text{Calories} = 4C + 4P + 9F.$$

(C = carbohydrate, P = protein, F = fat.)

2. Restriction of the protein content of the diet to 1·0 to 1·5 grammes per kilogram of body weight has already been mentioned.

3. Restriction of the carbohydrate and the balancing of the fat and the carbohydrate is universally accepted as a principle, but in detail the degree of carbohydrate restriction is at the present time one of the most debated points in diabetic treatment. A few years ago a diabetic diet was arranged in such a way that the relationship between the total glucose equivalent and fatty acid contents of the diet did not exceed the proportion of 1·0/1·6. This was indicated usually as G/F.A. The total glucose equivalent, which is known as the G or glucose value of the diet, is obtained by the formula:

$$G = 100\%\ C + 58\%\ P + 10\%\ F.$$

And the fatty acid:

$$F.A. = 90\%\ F + 42\%\ P.$$

Under this scheme the total glucose equivalent content of a diet rarely exceeded 120 grammes, and the amount of carbohydrate, as such, was seldom more than 70 or 80 grammes. Recently, as the result of much work both in Europe and in America, it has been found that the carbohydrate content of a diet can be considerably increased without ill effect to the patient, provided the fat is reduced *pari passu*. It is too early to expect dogmatic statements as to the relative merits of the two lines of treatment. White wisely states the position thus:

> The advantage of a diet which contains the carbohydrate equivalent of the normal diet, not more than half or even less than one-quarter of this amount, or which makes use of large amounts of protein or fat, rests upon an insecure foundation which the future, with its various possibilities, may clarify.

However, it is not my intention to enter into any controversy over this matter, and I shall merely state that there is a tendency to the use of larger amounts of carbohydrate than formerly, although from my own personal experience I am not satisfied that it is possible to use such diets in all cases.

So much for the principles underlying the dietary control of diabetes. I shall now proceed to the application of these to the treatment of the patient.

Confronted with an untreated diabetic patient, what procedure is to be adopted? Let me state that, if possible, such a patient should be placed in hospital for a short period. The reason for this procedure is that it is possible to get the patient under control in a much shorter space of time than if he attempts weighing his food and testing urine at home away from supervision and without proper instruction. For patients requiring insulin I would say that a short period of hospital instruction is essential. Surely, if a patient is expected to treat himself for the rest of his life, a short period of schooling in the proper methods is not wasted time.

Now, as to the question of diet. Before the days of insulin there was only one method of rendering a diabetic urine sugar-free, and it was not always successful. I mean by starvation. Now, if confronted with a patient who has lost one or two stone in weight and who is feeling tired and weak and looks wasted, there is no point in starving or in under-feeding such a patient. It only causes further loss of weight and makes the task of building up the weight a longer process. Feed the patient from the beginning of your treatment.

Consider the case of a young adult who, before the onset of diabetes, weighed 70 kilograms (eleven stone) and in the space of a few weeks has lost ten kilograms in weight. How much food will he require? Obviously his energy requirements must be based, not on his present weight, but on his ordinary weight, so that his calorie requirements will be at least 25 to 30 times 70, that is, 1,750 to 2,100 calories per day.

How is this to be made up? The protein value is easily set at 70 grammes per day (one gramme per kilogram of body weight). The carbohydrate and fat may vary within wide limits, always remembering the necessity of providing adequate carbohydrate for the metabolism of fat. For a diet of approximately 2,000 calories the values could range between:

Carbohydrate	50 to 275	
Protein	70 to 70	
Fat	170 to 70	
Calories	2,010	2,010

But it would be advisable to adopt a midway value and prescribe values such as:

Carbohydrate	120
Protein	70
Fat	140
Calories	2,020

How can such a diet be constructed? How is such a diet to be divided into various meals?

It is usual, in patients who are not receiving insulin or in those cases in which insulin is being taken three times per day, to divide the diet equally between the three meals, so that for breakfast, dinner and supper this diet would be split thus:

Carbohydrate	40·0
Protein	23·3
Fat	46·6

(See Table I.)

TABLE I.
Diabetic Diet (Evenly Divided).

	Carbohydrate (120).	Protein (70).	Fat (140).
Breakfast.			
Egg, 1		6·7	5·3
Bacon, 45 grammes (uncooked weight) (1½ ounces)		4·7	29·1
Milk, 180 grammes (6 ounces)	9·0	5·9	7·2
Oatmeal, 20 grammes (uncooked weight) (¾ ounce)	13·2	3·3	1·5
Uneeda, 1	5·0	0·5	0·5
15% Fruit, 105 grammes (3½ ounces)	13·3	0·8	0·5
Butter, 4 grammes (⅛ ounce)		0·4	3·4
	40·5	22·3	47·5
Dinner.			
Meat, 75 grammes (2½ ounces)		14·1	14·1
3% Vegetable, 150 grammes (5 ounces)	4·5	2·4	0·5
15% Fruit, 90 grammes (3 ounces)	11·4	0·7	0·5
Milk, 60 grammes (2 ounces)	3·0	2·0	2·4
Bread, 40 grammes (1½ ounce)	20·8	3·7	0·4
Butter, 34 grammes (1 ounce)		0·3	28·9
	39·7	23·2	46·8
Supper.			
Meat, 45 grammes (1½ ounces)		8·5	8·5
1·5% Vegetable, 120 grammes (4 ounces)	1·8	1·9	0·4
Cheese, 20 grammes (¾ ounce)	0·1	5·8	7·2
Milk, 180 grammes (6 ounces)	9·0	5·9	7·2
Bread, 30 grammes (1 ounce)	15·6	2·8	0·3
15% Fruit, 120 grammes (4 ounces)	15·2	1·0	0·6
Butter, 25 grammes (⅞ ounce)			21·25
	41·7	25·9	45·45
Carbohydrate = 120 grammes; Protein = 70 grammes; Fat = 140 grammes; Glucose = 175 grammes; Fatty acid = = 158 grammes; Calories = 2,020; Glucose/Fatty acid ratio = 1·1 : 1.			
Supper	41·7	25·9	45·45
Breakfast	40·5	22·3	47·5
Dinner	39·7	23·2	46·8
Total	121·9	71·4	139·75

Should the patient be taking insulin twice per day, that is, morning and night, it is often advisable to split the diet so that the bulk of the food coincides with the insulin injections, thus:

Carbohydrate	48	Two-fifths each for morning and evening meals.
Protein	32	
Fat	56	

And

Carbohydrate	24	One-fifth for the mid-day meal (see Table II).
Protein	16	
Fat	28	

Next, from one of the many books of food tables, an appropriate diet can be quantitatively built up, with a certain regard for the individual likes and dislikes of the patient. It is essential that all food should be accurately weighed and measured if you wish to stabilize the patient successfully.

A diet having been obtained for the patient, he should be tried on it for several days. If the urine shows no signs of becoming free of sugar, it is probable that insulin will be necessary, and it should be given without further delay. The initial dose varies with the severity of the case, but seven to ten units given about twenty minutes before the morning and evening meals is quite safe. The dose is increased or decreased until the urine is clear of sugar throughout the twenty-four hours.

It is not essential that the patient should remain in bed during this process of stabilization, and it is better if he is allowed to be up and about during this time. A careful record of the weight is kept, and if it is not increasing after the urine has been rendered sugar-free, an increase in diet is indicated. During the period in hospital the patient should receive elementary instruction in the nature and control of his condition and should be encouraged to read one of the reputable instruction books on the subject. He should become thoroughly conversant with the technique of urine testing. He should understand the care and use of a hypodermic syringe and be able to measure and inject his own insulin. He should receive elementary instructions as to personal hygiene (care of neck and feet) and the necessity for regular bowel action *et cetera*. He should receive, according to his own intelligence, some instruction in diet, and some member of his household should be taught the weighing and preparation of food. Substitution of various foods should be explained so that the diet can be varied from time to time, to avoid monotony, and accommodated to the various fruits *et cetera* in season during the year.

On discharge from hospital the patient should keep a careful record of the weight and urine testing, so that the progress can be easily followed. It is important to adjust the diet at such a value that the patient is able to do his work satisfactorily and yet maintain his weight at a normal figure.

TABLE II.
Diabetic Diet (Unevenly Divided).

	Carbohydrate (120).	Protein (70).	Fat (140).
Breakfast.			
Eggs, 2		13·4	10·5
Bacon, 30 grammes (1 ounce)		3·2	19·4
Milk, 180 grammes (6 ounces)	9·0	5·9	7·2
Oatmeal, 20 grammes (¾ ounce)	13·2	3·3	1·5
Bread, 30 grammes (1 ounce)	15·6	2·8	0·3
15% Fruit, 90 grammes (3 ounces)	11·4	0·7	0·5
Butter, 20 grammes (⅝ ounce)		0·2	17·0
	49·2	29·5	56·4
Dinner.			
Meat, 60 grammes (2 ounces)		11·3	11·3
3% Vegetable, 120 grammes (4 ounces)	3·6	1·9	0·4
Milk, 60 grammes (2 ounces)	3·0	2·0	2·4
15% Fruit, 90 grammes (3 ounces)	11·4	0·7	0·5
Uneeda, 1	5·0	0·5	0·5
Butter, 15 grammes (½ ounce)		0·15	12·75
	23·0	16·55	27·85
Supper.			
Meat, 60 grammes (2 ounces)		11·3	11·3
1·5% Vegetable, 120 grammes (4 ounces)	1·8	1·9	0·4
Bread, 30 grammes (1 ounce)	15·6	2·8	0·3
Milk, 240 grammes (8 ounces)	12·0	7·9	9·6
15% Fruit, 125 grammes (4 ounces)	15·8	1·0	0·6
Cream, 30 grammes (1 ounce)	0·9	0·7	12·0
Butter, 25 grammes (⅞ ounce)		0·25	21·25
	46·3	25·85	55·45
Supper	46·3	25·85	55·45
Breakfast	49·2	29·5	56·4
Dinner	23·0	16·55	27·85
Total	118·5	71·90	139·70

Another type of patient, the middle-aged obese female, is handled on slightly different lines. Far from increasing the weight by large diets, it is here necessary to reduce the weight to what would be considered normal for a person of her stated age and height. This involves deliberate under-feeding, not starvation, until such time as the weight approximates to normal. The rationale of such treatment must be explained to the patient and her cooperation obtained, otherwise she will often complain bitterly of lack of food and will tend to break diet. When the weight is sufficiently reduced the diet can then be increased so that the weight remains stationary. Such patients often become stabilized quite satisfactorily without insulin or, should insulin be necessary to render the urine sugar-free, it can sometimes be discontinued as the carbohydrate tolerance improves with loss of excessive weight.

For those who are unacquainted with the calculations necessary for construction of diets the line ration scheme of Lawrence is of considerable value. This is probably the simplest possible method of prescribing a balanced diet, and, read in conjunction with Lawrence's "A B C", is easily understood by the patient. It possesses the disadvantage that the ratio of fat and protein remains fixed, and for this reason it is difficult to apply it to certain types of cases, particularly to children.

After the first few weeks of diabetic *régime* most patients settle down to the new mode of life and food quite well. It is well for such patients, no matter how well they feel or how clear the urine tests, to report to their medical man at stated intervals, say, once every two or three months. In this way any slackness in the routine, any undue loss or gain in weight, and any difficulties in diet can be discovered and overcome.

Diabetes in Children.

Although fortunately rare, diabetes in children is, as a rule, severe. Without enlarging on this aspect of the subject, I would point out certain features peculiar to the diabetic child. First, the energy requirements and growth demand a higher caloric intake per kilogram than the 25 to 30 needed for an adult. Depending on its age, a child will require two, three, or even four times as much food per kilogram of body weight per day as an adult. The education of the diabetic child is largely a duty of the mother, and the careful training of the mother is the most important feature in the treatment of juvenile diabetes. The variable activities of the child, together with its relatively small glycogen storage as compared with the adult, make the stabilization of the diabetic child a difficult matter. It is sometimes almost impossible to keep its urine constantly sugar-free and yet avoid symptoms of insulin over-dosage. The mother must be trained to recognize these symptoms and be prepared to treat them promptly if they arise.

Insulin.

Insulin is administered by subcutaneous injection 15 to 30 minutes before a meal. Given orally, it has always proved useless. Depending upon the severity of the case, it is given once, twice, or thrice per day. As already stated, it is advisable to adjust the values of the various meals so that the larger meals coincide with the insulin injections.

Local allergic reactions occur rarely at the site of injection and generalized anaphylactic reactions are very uncommon. A change in the brand of insulin used is frequently sufficient to remedy this trouble.

Reactions from insulin over-dosage (hypoglycæmia) may occur from half to twelve hours after an injection of insulin and are most frequently seen from four to six hours later. Such reactions are most often seen during the process of stabilization. Those in the stabilized patient are most likely to occur following undue physical exertion or following carelessness in management of the diet.

The symptoms of hypoglycæmia arise because of an undue lowering of the blood sugar, and the initial symptoms may vary from case to case. Restlessness, tremor, weakness, sweating, emotional disturbances, faintness and epigastric pain are often the initial symptoms. More severe reactions are associated with motor or sensory disturbances, which may be followed by paralysis, unconsciousness and convulsions.

In the early stages of insulin reaction the symptoms are quickly relieved by the taking of some rapidly absorbed carbohydrate by mouth. It is a good rule to advise all patients taking insulin to carry at all times a lump or two of loaf sugar to take if such symptoms arise.

The severe reactions are treated by the giving of half to one ounce of glucose or cane sugar by mouth, if possible. If this cannot be done, one cubic centimetre of pituitrin should be given intramuscularly, or 10 to 15 cubic centimetres of a 50% solution of glucose should be given intravenously. It is important to realize that, no matter how alarming the symptoms of an isolated hypoglycæmia may be, there is little danger to life from the lowering of the blood sugar, and when this is corrected, such patients make a dramatic recovery.

Much might be said of the abuses of insulin. The patient who states that he does not want insulin usually suffers from numerous erroneous ideas as to the nature and the effects of it. If these are explained in a simple way, treatment can usually be instituted. On the other hand, insulin should never be used to allow a patient to overindulge his taste for carbohydrate or to commit other dietary indiscretions. It is not a single therapeutic measure, but an adjunct to correct dietary *régime*.

Complications.

Should a diabetic become ill from any cause, the important question of diet at this stage must be

considered. If, because of gastro-intestinal upset or pyrexia, or some other such cause, a patient is unable to continue with his ordinary diet, what is to be done? Many patients cease eating and, if taking insulin, omit their injections, often with disastrous results. If a patient is unable for any reason to take his stated diet, he should be encouraged to take the carbohydrate components of the diet, or equivalent substitutions thereof, and reduce or omit the fats and proteins (Table III). The reason for this is simple. When a person starves, he immediately draws on the body tissues (chiefly the fats and proteins) for the bodily needs. If a diabetic does this he is unable, from lack of available carbohydrate, completely to metabolize the fats, and symptoms of incomplete fat metabolism or ketosis develop. Therefore, carbohydrate and insulin are necessary.

In the presence of infection and fever insulin is often less effective than in normal circumstances and, as a rule, the insulin dosage has to be increased for the time being. The amount of increase is determined by the urine findings in each individual case from day to day. A diabetic who does not ordinarily require insulin may need it as a temporary measure during the height of an infection. If the patient is gravely ill, it is possible to convert the diet into a completely liquid form, so that it can be easily absorbed. During the recovery phase the insulin will be decreased and the patient gradually encouraged back on to ordinary diet again.

TABLE III.
Diabetic Liquid Diet for Diabetics after Operation and after Coma.

	Carbohydrate (110).	Protein (60).	Fat (60).
Breakfast.			
Gruel { Oatmeal, 20 grammes (uncooked weight) (¾ ounce)	13·2	3·3	1·5
Gruel { Milk, 120 grammes (4 ounces)	6·0	4·0	4·8
[1]Egg Nogg { Milk, 120 grammes (4 ounces)	6·0	4·0	4·8
[1]Egg Nogg { Egg, 1		6·7	5·3
Orange juice, 105 grammes (3½ ounces)	13·3	0·8	0·5
	38·5	18·8	16·9
Dinner.			
[1]Egg Nogg { Eggs, 2		13·4	10·5
[1]Egg Nogg { Milk, 120 grammes (4 ounces)	6·0	4·0	4·8
Coffee—milk, 120 grammes (4 ounces)	6·0	4·0	4·8
Orange juice, 180 grammes (6 ounces)	22·9	1·4	0·9
	34·9	22·8	21·0
Supper.			
Milk, 150 grammes (5 ounces)	7·5	5·0	6·0
Soup { Egg, 1		6·7	5·3
Soup { 1·5% Vegetable, 90 grammes puréed (3 ounces)	1·4	1·4	0·5
Soup { Cream, 15 grammes (½ ounce)	0·5	0·3	6·0
Soup { Milk, 120 grammes (4 ounces)	6·0	4·0	4·8
Orange juice, 150 grammes (5 ounces)	19·1	1·2	0·8
	34·5	18·6	23·4
Carbohydrate = 110 grammes; Protein = 60 grammes; Fat = 60 grammes; Glucose = 151 grammes; Fatty acid = 82 grammes; Calories = 1,220; Glucose/Fatty acid ratio = 1·8 : 1.			
Supper	34·5	18·6	23·4
Breakfast	38·5	18·8	16·9
Dinner	34·9	22·8	21·0
Total	107·9	60·2	61·3

[1]Egg noggs may have various flavours, for example, coffee, vanilla, marmite, lemon *et celera*.

Coma.

Unquestionably the most serious and the most urgent of all complications of the diabetic state, coma may be compared with rupture of an abdominal viscus. Every hour which elapses before treatment commences materially prejudices the chances of recovery. Diabetic coma arises because of the accumulation in the blood stream of intermediate products of fat metabolism, which appear because of the lack of available carbohydrate to complete the process. The predisposing factors are three in number, namely:

1. Of all cases of coma 70% occur because of carelessness and neglect of instructions as to diet and insulin. This is important, because with proper education many of these are preventible.

2. Infections complicating the diabetic state produce disturbance of carbohydrate metabolism and render insulin less effective. Reference has already been made to these cases.

3. Occasionally diabetes develops so rapidly that advice is sought for the first time when coma is imminent.

The onset of diabetic coma is usually gradual, its full development being a matter of some hours. The prodromal symptoms are numerous and may not all be found in any one patient. The commonest early symptoms are lack of energy, tiredness, anorexia, nausea, vomiting or retching, headache, vertigo. A little later nausea and vomiting become more marked; the tiredness increases to drowsiness, at first transient, but later becoming accentuated and deepening to actual coma. At this stage the patient may complain of severe pain, which may be located in any part of the abdomen or in the thorax. This may give rise to some difficulty in differential diagnosis. Examination of the urine will, however, give the clue to the underlying diabetic condition. The breathing at this stage may show some alteration; increased depth of respiration may be noted, later developing into the typical picture of air hunger.

The appearance of an established coma is well known. The patient lies unconscious, breathing deeply, the breath smelling of acetone; the skin is cold and clammy, the pulse thin and rapid, the temperature subnormal, the blood pressure low, the ocular tension greatly decreased, the tongue glazed and dry, and the urine is found to be loaded with sugar, diacetic acid and acetone.

The treatment of coma should be directed to the relief of ketosis and to the prevention of circulatory depression, which is frequently found in the later stages of this condition.

Insulin should be given in large doses, and often. Thirty to fifty units can be given at hourly intervals for several hours. If in doubt as to the blood sugar, it is advisable to commence the administra-

tion of glucose or some other easily absorbed carbohydrate quite early in the treatment, as it is necessary to provide an excess of available carbohydrate to relieve the ketosis. Thirty grammes of glucose to each injection of insulin should be sufficient. At the beginning of treatment it is often necessary to empty the bowel by an enema, and if vomiting is persistent a stomach wash-out is indicated. Every attempt must be made to maintain an adequate circulation by means of warmth and fluids given by mouth, by rectum, subcutaneously and, if necessary, intravenously. Blood pressure and pulse rate are the most useful guides to the state of the circulation. If there are signs of failure, with a rising pulse rate and a falling blood pressure, the intravenous administration of half to one pint of 1·8% saline solution is of great benefit. Stimulants, such as "Coramine", caffeine and strychnine, find a place in maintaining the general strength of the patient.

In the absence of blood sugar estimations frequent examinations of the urine must be made—at intervals of from two to three hours—as they provide a valuable guide to the progress of treatment. It may be necessary to catheterize the patient's bladder to obtain the necessary samples of urine. Decrease in the amount of diacetic acid, as shown by the ferric chloride test, is the first evidence that the ketosis is being overcome, and later the urine will be found to contain none. Acetone will probably remain for some time and disappears much more slowly. An examination of the urine for sugar must be performed on every specimen, and the absence of urinary sugar is a warning that the patient is in danger of hypoglycæmia.

The continuance of insulin therapy will depend upon the urinary findings and the general condition of the patient, more particularly in the decrease of air hunger and lessening of the depth of coma. It may be necessary to give some hundreds of units of insulin before much improvement is noticed. Intravenous administration of insulin is not such a satisfactory procedure as one would imagine. The effects are usually transient and of little benefit.

Following the recovery from coma, it is usually impossible to resume strict dietary control at once. For from twelve to twenty-four hours it is advisable to allow the patient to partake of easily assimilated carbohydrate and protein food, such as eggs, milk, Benger's food, arrowroot *et cetera*, and to allow fats to be taken sparingly. The insulin dosage at this stage will be determined by the urinary or blood sugar findings. Following this it is usually possible to start strict dietary control and to proceed to stabilization along ordinary lines.

Surgery in Diabetes.

Insulin has rendered possible many surgical procedures which were previously impossible because of the danger of coma. The preparation of the diabetic for operation involves one important principle—the avoidance of ketosis. Because of this it is well, if possible, to establish adequate reserves of carbohydrate. Large amounts of easily absorbed carbohydrate food, such as Benger's, gruel *et cetera*, should be given, together with insulin in the proportion of fifteen units to every thirty grammes (one ounce) of carbohydrate, for two or three days before operation.

The anæsthetic will vary with the operation to be performed. Local or spinal anæsthesia should be used if possible. Failing these, gas and oxygen, or ether given by the open method, may be employed. Chloroform should never be used because of its effects on the liver.

Following operation it is advisable to treat the patient along the lines suggested for the sick diabetic—mainly carbohydrate food in a fluid form with insulin varying with the needs of the case. It is advisable to try to get the urine sugar-free soon after operation, as an unstable condition of diabetes may interfere with healing.

Penalties of Neglect.

Having dealt with the question of rigid control of the diabetic state and having stressed its importance, the question arises: why be so strict? What does it matter if a patient has a little sugar in his urine? He feels none the worse for it and he feels none the better when it is absent. Provided he is kept out of the danger of coma, why do more?

Apart from the fact that a little latitude and indiscretion of diet pave the way for further indiscretions and cause a careless outlook in a patient, there is a second and far more important reason for careful control which is not so apparent.

In the pre-insulin days the diabetic patient lived comparatively few years and usually died from the effects of his disease. Since the introduction of insulin patients are living longer and there is now time to see remote secondary effects of the disease on other body systems. By far the most important of these effects is the tendency of the diabetic, no matter what his age, to develop arteriosclerosis of an unusual type, since it attacks muscular rather than elastic arteries. Such arteriosclerosis progresses insidiously and may give little or no evidence of its presence until it declares itself with dramatic suddenness. It is not associated with hypertension or with many of the symptoms of other forms of arteriosclerosis. It is at the present time one of the most serious problems of all in the control of diabetes. It is unquestionably more frequent and more serious in those patients who have neglected their treatment, and, although not at present preventible, it is much less likely to occur in the well controlled patient.

Confronted by a patient whose sight is failing from diabetic retinitis, or by one with gangrene threatening a leg, or yet by a patient crippled with a coronary infarct, it is well to reflect that by no

amount of rigid treatment at this late stage can one hope to repair the damage wrought by years of neglect.

Results of Treatment.

Insulin has been in use now for ten years, and the effects of the change it has wrought in diabetic treatment are to be considered. The death rate from diabetes in any part of the world has not decreased in the past decade. What, then, is the use of insulin? Owing to the standards adopted by statisticians, diabetes takes precedence over many other causes of death, and this explains in part the anomaly. Further, as insulin is not curative, but merely prolongs life, sooner or later every diabetic must die and be classified as having died from diabetes.

The results of insulin therapy are seen by studying the mortality in age decades. It is then at once apparent that the deaths of younger diabetics have decreased very considerably, and herein lies the true effect of insulin. During the past three years the Baker Institute has endeavoured, by a campaign of education of the profession and of the patient, to arouse interest in this disease and in its treatment. The figures of the diabetic mortality in Victoria during this period have shown a distinct alteration, which is significant that the scheme has been of some benefit to the community.

Reports of Cases.

POLYNEURITIC PSYCHOSIS IN PREGNANCY.

By ALLEN PRIOR,
Fifth Year Medical Student,
University of Sydney.

Clinical History.

MRS. C. was admitted to the Psychiatric Clinic at Broughton Hall in July of this year from the Crown Street Women's Hospital. At that time she was very much confused and had no appreciation of her position. Her history showed that she was four months pregnant. She had had pernicious vomiting and had been recommended for admission to the Crown Street Hospital for termination of the pregnancy, but this vomiting had yielded to medical treatment.

The examination of her mental condition showed that she was fully conscious. Her attention could be obtained, but she cooperated poorly in the examination. She had some illusions, but she denied any hallucinations. She exhibited poverty of ideas and was at times irrational. She had changing delusions of persecutory nature, such as the imminence of her being left naked on a beach by a party of foreigners and subsequently put into gaol. Her remote memory could not be tested, but her recent memory was definitely bad. She had no idea of the day and thought it was the early part of 1933; neither was she orientated as to place. She was very miserable and dull, as well as markedly labile, weeping copiously, and childishly emotional.

Her psychomotor activities were decreased. She lay in bed in a helpless state and made no attempt to aid herself. She was rather irritated by questions and carried on a disconnected conversation with herself, repeating phrases numberless times.

With regard to her habits, she was noisy at times and tended to be faulty. She was difficult with food.

On physical examination she was of early middle age, poor nutrition, slight build and pasty complexion. She bore the scar of a left temporal trephine. Her teeth and the angles of her mouth were covered with a collection of saliva. The abdomen bore well marked *striæ gravidarum*; the fundus of the uterus was just palpable at the *symphysis pubis*. The pulse rate was 120 per minute and has continued at this rate. The heart sounds were weak, but unaccompanied. The urine was acid and contained epithelial *débris*.

No disorder of speech function was present. The left pupil was larger than the right (post-traumatic); there was widening of the palpebral fissures and von Graefe's sign was positive; both pupils reacted to light and accommodation. There was slight tremor of the tongue.

Light touch was not impaired. Deep pressure gave rise to marked pain in the region of the posterior tibial compartment muscles. The scalp was also tender, but the remainder of the body was free from this manifestation. Direct pressure over the posterior tibial nerve and over the supratrochlear notch, the infraorbital foramen, the mandibular foramen and the facial nerve gave rise to very definite tenderness. Direct pressure over the ulnar nerve was not unduly appreciated.

Discrimination between heat and cold was impaired only in the lower limbs. Cold was always recognized, but in the lower limbs heat was called "cold". Her actions were perfectly well coordinated in the upper limbs, but she showed a loss of sense of position and of passive movement in the legs and was quite unable to walk, her legs sliding from under her. The knee jerk, the ankle jerk, the plantar reflex and the abdominal reflex were not elicited. The remainder of the systemic examination revealed no abnormality.

There were no known family tendencies, and the family history was normal. The patient had had an average schooling and after leaving school had worked as a clerk. She was married first fifteen years ago and lived with her husband for fifteen months until he was convicted of bigamy. The patient's son was then born and she returned to work as a clerk.

She remarried six years ago. For the past three years there had been financial troubles. Both husband and son are now out of work. The second marriage has been happy.

She has one living child by the first husband and has had four miscarriages by the second. The reputed cause of these miscarriages was pernicious vomiting. She had a trephine operation following an injury twenty years ago, and a uterine replacement, necessitated by the miscarriages, four years ago.

She is stated to have been a nervous child and to have walked and talked in her sleep until she was aged seventeen. She was of bright, cheerful disposition. There was credible evidence that alcohol was not a factor.

Since admission the patient has shown definite confabulation, telling of imaginary excursions and visitors. This she has now lost. She seems anxious to help, but is unable to on account of her memory loss. By reason of frequent questioning she has learnt by rote that she is in Broughton Hall, but even this lesson is not word-perfect and probably means nothing to her. She has a tendency towards frivolity and display. The loss of memory remains gross. The mental and physical symptoms are stationary.

The Wassermann and Kahn tests give no reaction. The urine shows no abnormal constituents. The blood urea is normal and microscopic blood examination reveals no abnormality.

Comment.

The pathology of polyneuritic psychosis is described by Moll[a] as consisting of degeneration, vacuolization and fibre loss, together with hæmorrhages into the basal ganglia.

Carmichael and Stern,[a] in a well studied series of cases, describe a degenerative, parenchymatous neuritis in peripheral nerves and in the spinal cord with consequent destruction of anterior horn cells. In the brain the

changes found were almost entirely confined to the cortex and were wholly incident on the cells. They describe an excessive deposit of normal lipochrome in abnormal situations and acute chromatolysis of cortical nerve cells.

The above authors quote Korsakoff's description of a case in which there were no changes in the cortex, but an increase in connective tissue in the column of Goll. He found parenchymatous degeneration of peripheral nerves, which was most marked distally. There was Wallerian degeneration in certain cranial nerves, being most marked in the vagus.

Clarke[2] quotes various authors showing changes in the metabolism of nerve cells, increase and thickening of vessels and thickening of their walls and fibre loss in various places.

In all of these cases, except that of Korsakoff, alcohol played a large part.

The ætiology of the condition is not a small matter. Carmichael and Stern state that Korsakoff recognized the syndrome in various fevers, in *diabetes mellitus*, in pernicious vomiting of pregnancy, and in cases of certain tumours. All this is in addition to alcohol, which is usually given as the main factor. The above authors quote Marcus as saying that in all alcoholic cases other disease processes are operating.

Ely[6] says that a multiple neuritis is common in pernicious vomiting of pregnancy and that it may occur without evidence of underlying infection. He further states that a mild psychosis of a Korsakoff type is very prone to occur in these cases. He quotes Hösslin to the effect that the *pro rata* incidence in pregnancy is greater than in alcoholism.

Henderson and Gillespie[3] state that the syndrome is not common and accounted for but 2% of alcoholic patients admitted to New York State hospitals in 1910.

Henderson[4] acknowledges the association between the syndrome and hyperemesis. He quotes a case of Eulenberg in which the psychosis developed after an abortion had been procured on account of hyperemesis.

The course in these cases is likely to be prolonged even under treatment. One woman, who was admitted with a psychosis of alcoholic origin and who developed manual as well as pedal paresis, could not stand and support herself before the end of a year. Henderson quotes a case described by Korsakoff in which recovery lasted over a period of four years.

Recovery would appear to begin after the supply of the main noxious influence is stopped. The length of time necessary before full function is restored must depend on the amount of damage done to the actual nerves involved. Since parenchymatous degeneration takes place in the peripheral nerves, the amount of regeneration will depend on how far this degeneration has progressed. Regeneration of nerve is ever a slow process, so that the earlier the syndrome is recognized, the less will be the actual nerve damage, the less the distance to regenerate and the earlier the prospects of recovery.

Degeneration is not entirely confined to the peripheral nerves. One case record shows that a woman admitted to hospital with Korsakoff's psychosis of alcoholic cause, very much exhausted and with marked tachycardia, died three weeks later after a period in which she had respiratory embarrassment and used entirely her accessory muscles. This, together with the tachycardia, points to degenerative processes being marked in the vagus and phrenic. In the vagus it was probably Wallerian degeneration as described by Korsakoff. Henderson quotes a similar case described by Polk.

With regard to further treatment of this case, therapeutic abortion must be seriously considered. The change that has occurred in her mental symptoms does not necessarily indicate a better prognosis. The polyneuritis is no less marked, and the memory defect has not improved, so that there is no guide to say whether the stopping of the hyperemesis has also put a stop to the cause of the damage of nervous tissue. Any indication of further attack, such as the development of polyneuritis in the upper limbs, would be of definite value, as would another attack of vomiting.

Acknowledgements.

I desire to acknowledge my very great thanks to Dr. Evans Jones, Superintendent of Broughton Hall, for permission to report this case and for access to clinical records and literature, also for his kind encouragement and advice.

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(3) Henderson and Gillespie: "Psychiatry", page 45.
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(5) Moll: "Korsakoff's Psychosis", *Journal of Mental Science*, 1915.
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Reviews.

DISEASES OF THE COLON, RECTUM AND ANUS.

WITH the vast clinical and scientific resources of the Mayo Clinic at its disposal, an experienced triad (Rankin, Bargen and Buie) has produced a work on "The Colon, Rectum and Anus", the perusal of which fills the reader with admiration and gratitude.[1] Though the text is written in a modest and scientific manner, the authors have no hesitation in expressing decided views as to many aspects of ætiology and treatment.

The book opens with short, well illustrated chapters on anatomy and physiology and on normal and anomalous development. Original work on the blood supply of the bowel by Rankin and Steward adds to existing knowledge. Congenital anomalies are invited to explain megacolon (Hirschsprung's disease), which, it is inferred, results from a spastic condition of a segment of colon. Hence the hopeful attitude towards operative measures on the sympathetic nervous system. Nature's method of fusion of proctodeum with the hind gut is held to be largely responsible for numerous infections of this region—cryptitis, papillitis—leading to ulcer, abscess *et cetera*. Diverticulosis and diverticulitis are usually confirmed by X ray diagnosis, as are, indeed, most of the diseases of the large bowel. Of 481 cases of diverticulitis only 48 were considered to require surgical treatment. Under the caption of "Colonic Manifestations of Systemic Origin" is placed "mucous" or "spastic" colitis. In 200 successive cases all were considered to be definitely neurotic. The details of treatment conform to this idea and are worthy of close attention.

Other diseases treated chiefly medically are the parasitic diseases, sprue, chronic ulcerative colitis *et cetera*. In the last named condition the authors are satisfied that the cause is usually a diplostreptococcus, and give experience of 647 patients, many of whom were treated by a special serum. In the treatment of various intestinal fistulæ stress is laid on the necessity of excluding the possibility of obstruction distal to the opening. Tuberculosis occupies the greater part of the chapter on granulomatous diseases. The rarer hyperplastic type, occurring, as it frequently does, in the ileo-caecal region, invites surgical attention, by excision if possible. Mention is made of Fraser's method of curing prolapse of the rectum by injections of absolute alcohol. The whole-hearted approval of Clubbe's work on intussusception is of special interest to the medical men of Australia. Benign tumours and polyposis are duly considered. The important subject of carcinoma of colon, rectum and anus is comprehensively dealt with. The pre-operative value of decompression and rehabilita-

[1] "The Colon, Rectum and Anus", by F. W. Rankin, B.A., M.A., M.D., F.A.C.S., J. A. Bargen, B.S., M.D., M.S., F.A.C.P., and L. A. Buie, B.A., M.D., F.A.C.S.; 1932. Philadelphia: W. B. Saunders Company; Melbourne, Brisbane and Christchurch: James Little and Son. Royal 8vo., pp. 846, with 435 illustrations. Price: 60s. net.

tion is emphasized. Selection of the best time for operation and of the best method of operation, the question of multiple stages of surgical treatment, the nature of the anæsthesia, and the routine employment of intraperitoneal vaccination are fully considered. The radical one- and two-stage operations of Miles, Coffey, Lahey, Jones, Rankin and others are fully described and comparison is made.

Radium and deep X ray therapy in the treatment of rectal cancer, with or without surgical measures, receive lengthy consideration. There is no doubt in the minds of the authors as to the great value of radium in curative treatment. Space prohibits enumeration of even a few of the many methods of application, ranging from the radium bomb to radon seeds. The injection treatment of hæmorrhoids follows the lines already published by Buie, the object being obliteration of the veins by sclerosis. The last chapter of the book is devoted to operative procedures.

With the exception of a few printer's errors, we have nothing but praise for this admirable book.

THE CARE OF THE FEET.

"FEET IN HEALTH AND DISEASE" by R. R. Hayhow has been written for the lay public "to enable readers to take reasonable care of their feet". This little book is interestingly written and the teaching is sound, but it contains too much for the average layman and too little for the medical man. We doubt whether it will ever reach the general public.

PROGRESS IN OBSTETRICS AND GYNÆCOLOGY.

THE third edition of "Recent Advances in Obstetrics and Gynæcology" is a welcome addition to the "Recent Advances" series.[2] This latest edition is the joint work of Aleck Bourne and Leslie Williams. It has been extensively revised and several new chapters have been added.

The first part is devoted to obstetrics and opens with an account of established practice in ante-natal care. There follows a general and statistical review of the causes of maternal mortality and of fœtal deaths. Chapters are devoted to *ante partum* hæmorrhage and the toxæmias of pregnancy. There is included also an account of the chemistry of the blood and urine in pregnancy and liver function tests. This part has been rewritten since the last edition by Dr. G. Roche Lynch. About fifty pages are devoted to an excellent account of recent views of the ætiology, bacteriology and clinical features of puerperal sepsis. An example of a fresh orientation is the emphasis placed upon the probable importance of carriers in the production of puerperal sepsis.

New in this edition is a chapter on anæsthetics, analgesics and narcotics in the conduct of labour. This will be a useful little chapter for the practitioner, for, though condensed, it is comprehensive and, as elsewhere in the book, references are given comprising the most important papers which have appeared in the last seven or eight years.

The Kielland forceps and their use are described in an appendix contributed by Dr. Kielland himself.

The section devoted to gynæcology comprises eight chapters. First is an outline of standard treatment of cancer of the cervix. The Paris and Stockholm techniques are given and there is a note of American views in regard to radium treatment. There follow a chapter on sterility and a well illustrated account of the surgery of prolapse of the uterus.

Endometrioma is allotted a chapter, and Sampson's and King's theories are impartially discussed. A brief account of the functional uterine hæmorrhages is given and, curiously, there are no references appended to this chapter, which is a new one in this edition.

The authors do their level best in twenty-five pages with the disorderly subject of the sex hormones. An outline of physiotherapy is contributed by Dr. Justina Wilson. This is purely a physician's account, and there is no mention of the surgical application of diathermy. Ten references are given, all German.

The concluding chapter is written by Dr. H. Courtney Gage on the uses of X rays in gynæcology and obstetrics, and sixteen X ray films are well reproduced.

"NERVES."

THAT a book has a large circulation is presumptive evidence of merit; "Outwitting Our Nerves", by Josephine A. Jackson and Helen M. Salisbury, falls within this category.[1] The book is based on the personal experience of Dr. Jackson, who practises psychiatry in California. She takes patients into her house, where they become members of her "family". "Outwitting Our Nerves" is a *résumé* of the advice she proffers, interspersed with illustrative case histories. The authors have distinct literary talent, the work is eminently readable and can be understood by the intelligent layman. The viewpoint is frankly Freudian, and Freud's principles receive both deference and full description; the latter is accomplished so skilfully that even a dyed-in-the-wool opponent of the school can hardly cavil at the impression which is created. At the same time, Dr. Jackson uses so much material gathered from the orthodox schools of suggestion and persuasion as to leave open the door for a criticism as to the reasonableness of her own particular bias, which is so strong as to cause apparently the repression of Adler from the field of psychiatric endeavour. Although a classical psychoanalysis is advocated in serious cases, it is apparent that a very large percentage of patients have been relieved on purely persuasive lines. The book is actually an excellent account of a persuasive technique in which Freud's work is used as the court of final appeal. The critic may be excused for wondering whether an equally forcible argument founded on other bases would not be equally efficacious.

"Outwitting Our Nerves" will be read with interest by members of the medical profession if for no better reason than to steal word pictures of utility in translating psychology into suitable terms for the benefit of patients. As an example of apt phraseology may be cited the following poem, which illustrates that too much thinking about and meddling with the viscera impairs the function:

> A centipede was happy quite
> Until a frog in fun
> Said, "Pray, which leg comes after which?"
> This raised her mind to such a pitch,
> She lay distracted in the ditch,
> Considering how to run.

Of particular value to the practitioner are the chapters headed: "That Tired Feeling", "That Interesting Insomnia", "In Which the Ban is Lifted—Dietary Taboos", "In Which We Relearn an Old Trick—The Bugaboo of Constipation", "In Which Handicaps are Dropped—A Woman's Ills", "The Female of the Species", and "Choosing Our Emotions". The captions are in themselves highly descriptive and help to create an atmosphere of interest.

That many of the cures seem to be too easy to be true may lead to moments of depression when one's own difficult cases are remembered; but that, after all, is to be expected. The author of a book on therapeutics for the layman must of necessity be both optimistic and dogmatic. A recital of failures would be as out of place as in a consulting room.

[1] "Feet in Health and Disease", by R. R. Hayhow, M.I.S.Ch.; 1933. London: John Bale, Sons and Danielsson, Limited. Crown 8vo., pp. 48, with illustrations.

[2] "Recent Advances in Obstetrics and Gynæcology", by Aleck Bourne, M.B., F.R.C.S., and Leslie Williams, M.D., F.R.C.S.; Third Edition; 1932. London: J. & A. Churchill. Demy 8vo., pp. 413, with 87 illustrations. Price: 12s. 6d. net.

[1] "Outwitting Our Nerves", by J. A. Jackson, M.D., and H. M. Salisbury; Second Edition, revised and enlarged by J. A. Jackson; 1933. Australia: Angus and Robertson, Limited. Demy 8vo., pp. 329. Price: 7s. 6d. net.

The Medical Journal of Australia

SATURDAY, DECEMBER 9, 1933.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

Reference to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE CONTROL OF MENTAL DEFECTIVES IN SOUTH AUSTRALIA.

In previous discussions in these pages on the subject of mental deficiency, emphasis has been laid on the preventive aspect. The student of preventive medicine, the psychologist and the psychiatrist rightly insist that people who are not responsible for their deviation from accepted social standards shall be treated for their mental defects. The benefit likely to result from the training of mental defectives will depend in large measure on the degree of deficiency displayed by them. As far as the higher grade of mental defectives are concerned, training may accomplish a great deal, and, provided they can be shielded from the greater stresses and strains of life, these persons, given a sympathetic environment, may be allowed to lead their own lives free from let and hindrance. When the mental defect is so great that training can have no effect, the afflicted person must be placed in such surroundings that asocial behaviour cannot take place. Most governments recognize this and make some sort of provision for the segregation of at least those mental defectives whose mentality is of the lower grade. The State, of course, is all powerful in this matter. In its wisdom, its rashness or its foolishness it may promulgate any law that it wishes; and the mental defective, even in these so-called enlightened days, may, when rashness or foolishness is displayed, be made to suffer unnecessarily. Mental defectives, in common with those who are afflicted with mental disease, are entitled to more than ordinary consideration, because they are unable to help themselves. The law makers sometimes forget this. They also sometimes forget that the administration of laws dealing with the care of mental defectives calls for special knowledge as well as for administrative ability.

In South Australia a retrograde step has been made. *The Mental Defectives Act*, 1913, has been amended, and the amendment does not reflect the wisdom of the law makers, nor is it likely to benefit the mental defective. *The Mental Defectives Act*, 1913, was administered by the Mental Defectives Board. The Board consisted of three members: the Inspector-General of Hospitals and two members appointed by the Governor. The Inspector-General of Hospitals, as was right and proper, was chairman of the Board. The two other members of the Board could be chosen from those whose special knowledge and training qualified them to deal with mental deficiency. With a board so constituted it is obvious that administrative problems, the psychiatric and psychological factors, and the economic aspect would be likely to receive proper consideration. The functions of the Board as set out in the Act were as follows:

Subject to anything prescribed, the Board shall have the control, management, and supervision of all institutions (except receiving wards, and also except such (if any) hospitals for criminal mental defectives as are situated in any prison, within the meaning of the "Prison Act, 1869"), and shall also have and exercise all such other powers and duties as are conferred or imposed upon it by or under this Act: Provided that the Board shall not have any power or duties as to the treatment, control, management, or supervision of the patients in any institution, except so far as expressly provided by this Act, or so far as their powers and duties as to the finances of an institution and the premises thereof may affect the patients therein. Notwithstanding anything contained in this Act, the Board, or any member of the Board authorized in that behalf by the Board, may make such inquiries as they or he thinks fit with respect to the care, treatment, or mental or bodily health of the patients in any institu-

tion, or with respect to any matter affecting the social or physical welfare, or comfort, or discipline of the patients in or inmates of any institution, or with respect to any other matter prescribed, and for the purpose of any such inquiry may visit and inspect any institution and may ask questions of the superintendent of any institution or of any other officer or person employed in any institution.

A bill to amend *The Mental Defectives Act*, 1913, was recently brought before the South Australian Parliament. According to the provisions of this bill, the Mental Defectives Board was to be abolished and the whole administration of the Act was to be placed in the hands of the Inspector-General of Hospitals. The Council of the South Australian Branch of the British Medical Association recognized at once that such a step would be a mistake. It made a protest to the Government and asked the Minister to delay final action until the Branch should have an opportunity of submitting a report on the matter. The Council made it quite clear that it had every confidence in the Inspector-General of Hospitals and that its action was in no way an attack on him. The representations of the Branch Council were ignored and the amending Act was passed. The haste of the South Australian Government in this matter is to be deplored. The Council of the South Australian Branch of the British Medical Association was obviously concerned only with the proper care and treatment of mental defectives. It is the only organized body of the medical profession in South Australia that could have expressed an opinion on what was evidently a medical problem. The Government missed an opportunity of displaying wisdom. Though the Act has been passed, we hope that the Branch Council will not let the matter drop. Acts of Parliament have been repealed before this. The Government might be made to see that it had made a mistake. Were it courageous enough to acknowledge its mistake, it would rise in the estimation of all thinking people.

Current Comment.

BACTERIAL ALLERGY.

THE separation and definition of anaphylaxis and immunity has been attempted by most writers on allergy; but because a hypersusceptibility to a foreign bacterial protein usually occurs in both conditions, allowance must be made for considerable overlapping of the two conceptions entailed. Arnold Rice Roth, in commencing a symposium on allergy at the Third International Pædiatric Congress, London, 1933,[1] recommends that anyone who wishes to discuss any aspect of allergy should make it clear at the outset, both to his hearers and to himself, just what limitations he places on the term; with this the majority of physicians will heartily agree. Pirquet, who created the term, included even the bodily changes which occur with old age and which favour superimposed malignant disease. Roth defines allergy in the much more restricted sense of acquired hypersensitivity. He deals in his contribution solely with the relationship of this hypersensitive state with that of immunity to infection. He reserves the term anaphylaxis for a type of reaction engendered by the spasmodic contraction of the smooth muscle of bronchi or blood vessels, rather than the necrotizing inflammatory effects of the allergic reaction. Both are fundamentally the same and represent the results of interaction of the sensitizing antigen with an antibody specific in each case for the particular antigen. When the allergic necrotizing inflammatory type of hypersensitivity is produced experimentally by the injection of foreign protein into the tissues, passive transfer of sensitivity can readily be effected. But when the sensitivity occurs as a result of the liberation of bacterial protein into the tissues during infection, it is ordinarily impossible to demonstrate the sensitizing antibody in the blood stream, and the sensitivity cannot be transferred passively to the normal animal. Professor Rich has, however, shown by tissue culture that the washed individual cells of an allergic body are themselves hypersensitive to bacterial antigen. He states that it is clear from his photographs that the washed cells of an allergic tuberculous body are readily killed by amounts of tuberculin which do not affect the normal. This author further holds the view that an allergic reaction is not necessary for the operation of immunity and that the two states are not directly proportionate. Experimental work pointing towards the differentiation of allergy from immunity and to the determination of the efficiency of immunity in the absence of allergy was undertaken in Professor Rich's laboratories and seems to show that such a separation is possible. Allergic inflammation, he holds, is not at all necessary for the more efficient destruction of bacteria in the immune body. The most striking characteristic of acquired immunity from which allergy has been excluded, is the remarkable indifference of the tissues to the bacteria which are being destroyed, which is exactly the state of affairs obtaining in natural immunity. Immobilization of bacteria, generally believed to be the result of a local allergic reaction, is just as definite in the immune non-allergic body as in the allergic one.

[1] *The Lancet*, September 2, 1933.

Rich therefore seems to have shown consistently that neither of the two fundamental protective processes of the immune body, namely, the prevention of the spread of bacteria and their efficient destruction, is dependent upon the presence of allergic inflammation.

The opposite side of the picture, namely, the development of allergy in the absence of immunity, has been obtained by Sabin, Smithburn and Geiger at the Rockefeller Institute. A hypersensitive state to old tuberculin has been produced by the repeated injection of purified tuberculo-protein, which carried with it no increased resistance whatever to the effects of the tubercle bacillus. In fact, the lesions appeared to be more pronounced in these animals than in the controls. Hypersensitiveness, then, to bacterial protein can be readily established, but is not even a measure of the degree of immunity, according to Rich. Support for this conception is reflected clinically in the negro, who has a high degree of sensitivity to tuberculosis, but a notoriously low resistance, and who lacks the ability to develop an immunity resembling that of the white man.

This striking summary of the progress of experimental work on allergy is both convincing and provocative. The general tendency till now has unquestionably been to regard allergy and immunity as mutually proportionate, especially in regard to tuberculosis, which has been studied more closely in this manner than any other disease. In fact, the latest classification of the pulmonary form of the disease is based upon its supposed allergic phases. It is obvious from the work of Rich and his collaborators that we must not use the tuberculin and other similar allergic reactions as an exact index of the measure of immunity. The bulk of clinical experience, however, would support the general conclusion that in the human being skin sensitivity in tuberculosis, scarlet fever and diphtheria at least can be used as an approximate guide to susceptibility. It is interesting to note also the application of the method of tissue culture to other problems than that of cancer. So far this work has been mainly applied to the laboratory animal. We must wait for its final lessons until it has been applied to man. There is no doubt that a huge field of interesting and profitable investigation has been exposed, which will probably modify to a vast degree our former conceptions of the relationships of bacterial immunity to bacterial allergy. Professor Rich seems already to have dealt the death blow to the dogma that bacterial allergy is itself beneficial and protective.

ANGINA PECTORIS.

THE view is now widely held that precordial pain produced by effort is due to the inefficiency of narrowed coronary vessels; but there is doubt whether this or aortic spasm or some other abnormality is responsible. *Angina pectoris* is an unfortunate term, usually signifying a terribly severe paroxysm of cardiac pain; but pain of cardiac origin may be of all grades of severity and be felt over areas of variable extent; coronary occlusion, of course, is manifested by violent precordial pain that is not known as *angina pectoris*. Considering the possibilities of confusion and the doubt concering the mechanism responsible for *angina pectoris*, or precordial pain of cardiac origin, it is not surprising that little progress has been made in the methods devised for the relief of this symptom. An interesting study of the comparative value of drugs used in the continuous treatment of *angina pectoris* has recently been made by William Evans and Clifford Hoyle.[1] These workers investigated the effects of various drugs on 90 patients during a period of two and a half years. Great care was exercised in the diagnosis, and persons with syphilitic stigmata were not included in the series. Seventy-three of the patients were males and seventeen females; thirty were of the Jewish race. The following drugs were tested: sodium nitrite, mannitol hexanitrate, erythrol tetranitrate, potassium iodide, "Luminal", chloral, morphine, papaverine, phenacetin, "Diuretin", euphyllin, belladonna, digitalis, "Lacarnol" and "Harmol". As a control they employed a *placebo* consisting of a moderate dose of sodium bicarbonate in infusion of gentian, sometimes coloured with a little carmine. The *placebo* was substituted for the active drugs at regular intervals. The administration of each drug save erythrol tetranitrate was accompanied by improvement in a small number of the cases in which it was tested; but the administration of the *placebo* seemed to have a similar effect. Evans and Hoyle remark: "Though scarcely convincing, there was some reason to think that chloral, morphine, papaverine, and phenacetin had a trifling influence in controlling the group incidence and severity of attacks." They were, however, unable to convince themselves that any drug tested by them was worthy even of trial in the routine treatment of the disorder; they therefore urge the necessity of studying the application of the general measures known to control the paroxysms and of promoting "the wider use of vasodilators, such as trinitrin, which are so often successful in the palliative treatment or even in the prevention of particular attacks". There are many possible fallacies in the interpretation of the results of experiments in the treatment of *angina pectoris*. Evans and Hoyle seem to have eliminated most sources of error; but they still, of course, had to depend for their information largely on the patient's own impressions, which are not always reliable. There was no death during the period occupied by their investigations; this fact alone is sufficient encouragement for further study.

[1] *The Quarterly Journal of Medicine*, July, 1933.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Examination of Pathological Tissue by Filtered Ultra-Violet Radiation.

C. J. SUTRO AND M. S. BURMAN (*Archives of Pathology*, September, 1933) have used ultra-violet radiation in the examination of pathological tissue obtained from more than 500 specimens. The examination of any tissue or organ by filtered ultra-violet radiation resolves that tissue or organ into its constituent elements. It is a macroscopic method of differentiating the various components of a specimen through variation in fluorescence. As the fluorescence probably depends on the chemical constituents in the nuclei, cytoplasm and intercellular substance of the tissues, the authors have been unable to find a specific correlation between the fluorescent reaction and the pathological diagnosis. Tissue of different kinds may sometimes emit approximately the same fluorescence. However, after experience, a particular type of tissue may usually be recognized with ease, and minor variations may be readily interpreted. One of the illustrations to the authors' article depicts an adenocarcinoma of the breast. To the unaided vision the tumour appears uniformly white. Under filtered ultra-violet radiation the area of the tumour is definitely divided into two portions. The upper, hazy blue area is medullary adenocarcinoma; the lower area is white and is scirrhous. Below this is a smaller blue area due to foci of carcinoma metastases. The authors used a mercury vapour arc lamp and filtered the radiation through a nickel oxide filter. The examination of the specimens must be conducted either in a dark room or in a dark hood.

Osteochondritis of the Head of the Femur.

L. J. MILTNER AND C. H. HU (*Archives of Surgery*, October, 1933), writing from Peiping Union Medical College, China, report an experimental study of the cause of osteochondritis of the head of the femur. They point out that this condition is also known as Legg-Calvé-Perthes's disease, *osteochondritis deformans juvenilis coxa*, and pseudocoxalgia. They experimented on dogs and rabbits. Their experiments were divided into three groups. In the first they injected alcohol into the periosteum of the neck of the femur. In the second group the periosteum and the reflected portion of the synovial membrane were stripped back from the chondro-osseous margins of the head to the base of the neck. In addition, alcohol was injected into the periosteum in order to increase the amount of fibrosis and to delay the formation of a new blood supply. In the third group, in addition to carrying out the procedures described for the second group, the authors tied the round ligament with a silk suture. In the third group alone were constant changes produced. In all the animals except one of this group gross examination revealed roughening and slight flattening of the head. In this exceptional instance microscopic examination revealed necrosis of the bone cells of the head and of the marrow. In all the other cases of this group aseptic necrosis of the bone was present. The authors point out that the changes produced by them are similar to those found in osteochondritis of the head of the femur in human beings.

Hetero-Transfer of Filtrable Tumours.

W. J. PURDY (*The British Journal of Experimental Pathology*, August, 1933) reports an investigation by means of immune serum into the hetero-transfer of two filtrable tumours, the Fujinami myxosarcoma and the Rous sarcoma Number 1. He refers to the conclusion reached by himself and Gye in 1931, that the species-specific element, present in an active tumour filtrate and necessary for infectivity, must be derived from the tumour cells themselves and not from the normal tissues of the host. He also refers to conclusions of his own regarding the tumours in question: (i) that when minced fowl-grown Fujinami myxosarcoma is injected into an adult duck, the new tumour is formed chiefly or wholly by multiplication of infected host cells; (ii) that when minced fowl-grown Rous sarcoma Number 1 is injected into a duckling, the new tumour that arises is formed solely by multiplication of fowl cells from the inoculum; (iii) that when minced fowl-grown Fujinami myxosarcoma is injected into a duckling, the new tumour that arises is formed in part by cells in the inoculum and in part by infected cells of the host. The author has carried out a series of experiments to test the truth of these conclusions. His method of analysis is a serological one. It is based on the observation that in the presence of complement an appropriate anti-embryo immune serum will neutralize the infective agent of a tumour filtrate. The author gives three examples of his experiments and concludes that his previous observations have been confirmed.

Arterial Thrombosis.

G. SCHERR (*Deutsche Medizinische Wochenschrift*, June 16, 1933) writes that arteriosclerosis is the first causal factor of arterial thrombosis and that this is shown by its occurrence in the later years of life. In the vast majority of cases widespread vessel sclerosis is present. In young people arterial thrombosis occurs on a syphilitic basis and the author found four such cases in twenty-five autopsies. It is well known that industrial poisons, especially lead, may lead to vessel damage and secondarily lead to the formation of autochthonous thrombosis, but this is difficult to prove. Statistics and life insurance experiences prove that during the last few years there has been an increase in the number of cases of arterial thrombosis.

Mucus and Bacterial Invasion of the Intestinal Mucosa.

H. W. FLOREY (*The Journal of Pathology and Bacteriology*, September, 1933) has made some observations on the functions of mucus in regard to the early stages of bacterial invasion of the intestinal mucosa. The author made observations of the intestinal mucosa of a decerebrated cat and also administered hydrokollag to a cat that had been starved for twenty-four hours. He concluded that mucous secretion has as one of its principal functions the cleansing of the villi from small adherent particles. This cleansing mechanism plays a considerable rôle in preventing virulent bacteria from staying in any one place sufficiently long to damage the epithelial cells. The mucus mechanism is by no means perfect. The author tried to discover the first steps of an intestinal infection. He describes experiments in which he used *Bacillus aertrycke*, and reproduces drawings of organisms in contact with the epithelial cells and in the cells. It is uncertain how the bacteria gain entrance to the cells, but the author holds that it is not through phagocytic activity. Bacteria that cause lesions slowly must be lodged in some parts of the intestine for a considerable time before penetration following epithelial damage can occur.

The Relation of Hepatitis to Cholecystitis.

J. F. NOBLE (*The American Journal of Pathology*, July, 1933) discusses the relation of hepatitis to cholecystitis. He points out that cholecystitis is often regarded as being preceded by hepatitis. He has examined the liver in a series of 212 unselected but not consecutive autopsies. In this series the portal spaces were free from inflammatory cells in only two instances; in three other cases the reaction was practically negligible. This portal infiltration seemed to have no particular relation to generalized infections or to any specific type of disease. It bore no relation to the non-clinical cholecystitis frequently found in routine *post mortem* examinations. The author points out that the cholecystitis found in routine *post mortem* examinations is seen less frequently in the female than clinical cholecystitis and is more frequent in the younger age groups than clinical cholecystitis. He also found that cirrhosis occurred too rarely to justify the conclusion that the hepatitis described by him has any ætiological relationship to cirrhosis.

MORPHOLOGY.

The Spinal Parasympathetic.

KEN KURA *et alii* (*Quarterly Journal of Experimental Physiology*, June, 1933) state that both greater and

lesser splanchnics contain vasodilator fibres for the kidney. Stimulation of these nerves causes vasodilatation when the sympathetic vasoconstrictor fibres are blocked by nicotine applied to the cœliac and renal ganglia and to the kidney hilum. This vasodilator effect, they hold, must be due to spinal parasympathetic fibres in the splanchnics. They also state that sympathetic vasoconstrictor fibres must also be contained in both the greater and lesser splanchnics, because section of either nerve evokes noticeable diuresis, and diminution of kidney volume is produced by their stimulation. In another article of the same date they conclude that excitation of the spinal parasympathetic fibres promotes the internal secretion of the pancreas.

The Pericapillary Cells in the Mesenteries of Rabbits.

J. B. ROGERS (*The Anatomical Record*, September, 1932) confirms the report that capillary contraction is rare and quite negligible in effect on mammalian circulation. He demonstrates that stimulation of cells adjacent to capillaries did not alter the flow of blood or the outline of the capillary. Also he was unable to demonstrate the presence of myofibrils in the adventitial cells adjacent to the capillaries, as has been demonstrated in the nictitating membrane of frogs.

The Anterior Pituitary of the Albino Rat.

H. A. CHARIPPER AND H. O. HATERIUS (*The Anatomical Record*, September, 1932) state that the anterior pituitary of the female albino rat undergoes a definite cyclic rhythm in predominant cell type present, as revealed histologically. This rhythm can be correlated with the phase of the sex cycle present when the gland is obtained. During œstrus the anterior lobe is found to be predominantly basophilic; during the diœstrus interval it presents an eosinophilic picture. In instances of experimentally induced or of spontaneous continuous œstrus the *pars anterior* is characterized by a pronounced basophilia, essentially similar to that occurring during normal œstrus in the cyclic animal.

The Posterior Longitudinal Bundle in Man.

M. F. LUCAS KEENE AND E. E. HEWER (*Journal of Anatomy*, July, 1933) give a brief account of the constitution of the posterior longitudinal bundle as given by different writers, and describe their own investigation, based on serial sections of the brain stem of specimens ranging from ten-millimetre embryos to the nine-month infant. The posterior longitudinal bundle appears first in the twelve-millimetre embryo. It is one of the earliest to be myelinated, the process beginning at about fourteen weeks. In the early weeks of development (previous to eight and a half weeks) there are two unconnected groups of fibres, namely, a group connected with Deiter's nucleus, which extends up into the lower part of the pons and downwards into the medulla and spinal cord, and a group found in the upper pons region, extending up to the fourth nerve nucleus. At fourteen weeks myelinated connexions of the bundle are seen with the nuclei of Deiter and of the sixth and fourth nerves. At twenty-four weeks myelinated connexions occur with the nuclei of the third nerve and the posterior commissure. At about twenty-eight weeks a few myelinated fibres are seen crossing the mid-line in the supramammillary region. Their destination is uncertain, and they continue to increase in numbers in the older specimens, though all do not cross. The nucleus of the posterior commissure makes a definite connexion with the posterior longitudinal bundle and furnishes a large contingent of fibres to the ventral part of the posterior commissure. In the region of the nuclei described by Darschewitsch and Cajal four nuclei are identified and described.

The Blood Supply of the Lateral Geniculate Body.

A. A. ABBIE (*Journal of Anatomy*, July, 1933) has studied the blood supply of the lateral geniculate body in a representative phylogenetic series including six anthropoid, two lemur, four sheep, two marsupial, two crocodile and two sphenodon hemispheres, as well as human material. As far as the human material is concerned, he concludes that the anterior chorioidal artery plays a large and constant part in the blood supply of the lateral geniculate body. This artery is related to the lateral aspect of the ganglion, while the posterior cerebral branches are distributed more medially and posteriorly. The area of greatest functional importance—the macular projection area—has by far the richest blood supply, and this supply is drawn from the double source of the anterior chorioidal and posterior cerebral arteries. He brings forward evidence to show that the end arteries within the lateral geniculate body are distributed in close relationship with the projection of the visual fields in this ganglion.

The Medial Geniculate Body and the Nucleus Isthmi.

W. E. LE GROS CLARK (*Journal of Anatomy*, July, 1933) states that it has been held by many authorities, on the basis of comparative anatomical studies, that the medial geniculate body is rather to be regarded as a mesencephalic element which has become secondarily approximated to the diencephalon during the course of evolutionary differentiation of the forebrain. In this present communication he gives an account of an investigation, one of the chief aims of which is to consider this interpretation. He concludes that the main part of the medial geniculate body of mammals is developed as a caudoventral extension of the main sensory nucleus of the thalamus, drawn out, it would appear, under the neurobiotactic influence of auditory impulses which pass up to it from caudal levels. A study of the brains of primitive mammals in which the tectum is large shows that the *nucleus isthmi* of reptiles is also present and well developed in these forms, and is represented by a part of what has usually been called the dorsal nucleus of the lateral fillet. In higher mammals the nucleus is much less obtrusive in association with the diminished importance of mesencephalic centres generally.

The Sexual Cycle in the Human Female.

GEORGE N. PAPANICOLAOU (*The American Journal of Anatomy*, May, 1933) gives a review of the literature on cyclic changes in the vaginal fluid as revealed by the vaginal smear method, and furnishes additional material toward a clearer understanding of the human cyclic changes. He finds that the various stages are less typically expressed than in some of the lower mammals and are subject to greater variability. This investigation is based on daily smears taken from a group of selected normal women and numerous cases of pregnancy, of *post partum* and of various pathological conditions. The female sex cycle was divided into four phases and one stage: (*a*) the menstrual phase (first to seventh day), (*b*) the copulative phase (eighth to twelfth day), (*c*) the ovulative stage (twelfth to thirteenth day in most typical cases), (*d*) the proliferative phase (seventeenth day up to next menstruation). This classification was based on comparative considerations permitting its application to man as well as to other mammals. During the copulative phase in typical cases the vaginal smears are characterized by a more or less complete leucopenia and the epithelial cells show an increasing tendency to cornification. The cornified cells are generally nucleated. The ovulative smear is characterized by sudden increase in the number of leucocytes. The onset of pregnancy is marked by definite vaginal smear changes, and after parturition the smear undergoes a typical modification. The condition of the ovaries in relation to the various vaginal smear stages has been studied in seventeen cases operated upon by Dr. Ward. The study and analysis of all these cases offers evidence in favour of the view that ovulation normally occurs near the twelfth or thirteenth day, but that an earlier appearance (ninth to tenth day), or a long delay, or even a suppression, may eventually occur. In the normal cases, in which the onset of ovulation has been evidenced by corresponding changes in the vaginal smears, the most frequent time of ovulation was found to be between the twelfth and thirteenth days (range seven to seventeen days).

Special Articles on Treatment.

(Contributed by request.)

XXII.

THE TREATMENT OF CONJUNCTIVITIS.

CONJUNCTIVITIS is, as the name implies, an inflammation of the modified mucous membrane that lines the lids and covers the eyeball, and its treatment is directed towards: (i) Relieving the symptoms, which resemble those connected with inflammation of mucous membranes elsewhere in the body; (ii) arresting and curing the morbid process which may lead to damage of the affected tissue, or the adjacent cornea, and give rise to unfortunate sequelæ, such as scarring and contraction of the conjunctiva, alteration in the alignment of the lid margin and lashes, or corneal nebulæ through the occurrence of secondary corneal ulcers.

It occurs in various forms, which may be divided conveniently into acute, subacute and chronic.

Acute Conjunctivitis.

In the incipient stages of a developing acute attack, and in the hyperæmic stages of such burns and irritations as occur in snow blindness and with electric arc flashes, cold applications may be soothing and are best obtained by laying pieces of lint about the size of an eye shield on a small block of ice, placing them when cold on the affected eye or eyes and returning them again, when warmed up, to the block for recooling. Once the discharge has started, warm applications are indicated if the degree of discomfort suggests it; but the important principle of permitting free drainage of the discharge must be observed, and this requires as little interference with the opening of the lid as possible, so that all bandaging and compresses of any weight must be eschewed, although a loose dressing may be laid across the eyes, or, to shut out the light, a shield of brown paper may be fashioned. This indicates that when a monocular infection is present and it is desired to protect the uninjured eye, it is that eye which is covered, or sealed off, and not the affected eye. If the swelling be intense, the upper lid may extend over the lower and the patient be unable to raise it in the ordinary way and it must be lifted by the finger to permit of the escape of muco-pus. Particularly in the swelling met with in *ophthalmia neonatorum* intramuscular injections of whole milk boiled for four minutes, in doses varying from one to two cubic centimetres, may prove valuable. These may be given on the first, second, fourth and fifth days. In the case of adults large doses of six cubic centimetres may be given, but too large and too frequently repeated injections may give rise to severe abdominal pains. In place of milk "Aolan" may be given in corresponding amounts. The pain in the eye may tend to a further aggravating spasm of the lids, and some general sedative may be advisable or strips of adhesive plaster may be so applied as to drag the upper lid slightly upwards to the forehead and the lower lid downwards to the cheek. In addition to promoting a free passage for the discharge, the eyes should be kept as clean as possible by gently flushing out the eye with some innocuous fluid, such as normal saline solution, weak Condy's fluid (just coloured), boracic lotion, *et cetera*, but the washing out must be done gently, as too vigorous measures may damage the epithelium of the cornea, which tends to become softened. The frequency of the washing out will depend on the rapidity with which the discharge forms. Care in thus cleansing must be scrupulously observed in cases of gonorrhœal infection, as it is more than probable that many of the disastrous corneal ulcers that tend to appear in adult cases are traumatic in origin.

In the attempt to combat the infective agent various chemical remedies are employed, but their efficiency is not simply a factor of their calculated antiseptic qualities as observed in the laboratory. Argyrol in 10% solution is but a mild antiseptic, and yet it is in general use as a successful remedy, both in acute and chronic cases, but patients should be warned that it cannot be used indefinitely as a stock household remedy on account of the argyrosis which appears after prolonged use, and they should also be informed that it stains clothing, bed-clothes *et cetera* indelibly. In acute cases with profuse discharge as in *ophthalmia neonatorum* it may be used in strengths up to 30% or 40%, but as the amount of the muco-pus diminishes, the percentage must be reduced, and when there is no discharge it should be stopped. It is instilled at intervals which shorten in proportion to the severity of the attack. Neo-silvol, which is not so conspicuous in colour, may be used in its place. If the dark coloured protargol is used, its strength should be half that of the corresponding argyrol solution. These drops are comparatively painless, but if a case is proving obstinate silver nitrate in 0·5% to 2% solution may be necessary as an application to the everted lid surfaces once a day, any excess being neutralized and washed off with normal saline solution. The 0·5% solution smarts, the 1% smarts more distinctly, and the 2% may be decidedly painful. Part of its usefulness results from the superficial desquamation of infected cells that follows its use. Where there is a tendency to sloughing as in membranous conjunctivitis it should not be used, nor should it be given for home use. The more recent mercurochrome in 1% to 2% solution, used two-hourly if necessary, is also a very useful drop, and like acriflavine (one in 2,000) is usually not uncomfortable. The staining of the surrounding skin is lessened by vaselining the parts. *Lotio hydrargyri perchloridi* is a safe and useful home remedy, but the one in 5,000 solution as a rule causes too much discomfort. One in 15,000 to one in 20,000 is sufficiently strong and not painful. In later stages, where there is little or no discharge, astringents are indicated, and a solution of zinc sulphate 0·25% to 1%, aluminium sulphate 1%, or tannic acid 1% will get rid of the residual hyperæmia and enlarged follicles as in gonorrhœal ophthalmia.

If the diagnosis of a special type is established, specific remedies may be used, for example, "Optochin" 1% to 2% in the pneumococcal form, antiserum in the diphtheritic, zinc sulphate in the angular, but the "Optochin" is scarcely suitable for use in general practice, as it may cause a pronounced reaction.

Before passing from the consideration of acute infectious cases, it is well to emphasize again that in gonorrhœal ophthalmia: (i) Milk injections are of first importance, so much so that in the Children's Hospital in Perth it has been an established first line of treatment for the past ten years, and that (ii) gentleness in manipulating the lids is absolutely essential.

In heat or light ray burns and snow blindness soothing applications are more required than antiseptics, and here, in addition to ice externally, a weak cocaine drop 0·25% may be used if the eyes are kept closed. "Scarlet red" ointment 2% may be used to promote epithelialization of the desquamated areas.

Subacute Conjunctivitis.

The subacute forms may be treated on the foregoing lines, but the presence of foreign bodies and dacryocystitis, or in babies lachrymal obstruction, must be excluded.

Chronic Conjunctivitis.

In chronic conjunctivitis treatment by astringents, that is, zinc sulphate, as above mentioned, is mainly indicated, although the more active silver nitrate (half a grain to the ounce) may be indicated; but the chronicity may have led to a chronic catarrhal condition of the Meibomian glands and it may be necessary to unload them by massage before the lid condition will improve. The prolonged inflammation of the lower lid may also lead, partly through

the thickening and partly through atony of the lid, to a commencing ectropion, and treatment should, therefore, be continued until the conjunctival swelling is reduced at least as far as possible. The ectropion may in the aged increase to such a degree that it can be cured only by a plastic operation. Short of that, epiphora, with all its inconvenience, may be caused from the eversion of the punctum of the lower lid.

If the skin of the lid is irritated by constant discharge or watering, some ointment should be used for protection, for example, white vaseline, *unguentum ichthyol compositum*, zinc ointment, especially in the presence of angular conjunctivitis. Goggles to protect from glare and wind and dust are comforting and beneficial, and if there are errors of refraction their correction is advisable and may be necessary.

The special form known as trachoma must be dealt with separately. In it thoroughness in treatment is essential. I do not mention treatment of the so-called acute trachoma as the remedies for acute conjunctivitis are applicable to the mixed infection, but for the true chronic disease it may be stated that home treatment by drops is not sufficient. To advise such treatment, except when the condition is well under control and obviously resolving, is to do the patient a disservice, as, apart from the unusual cases that undergo degeneration and spontaneous cure, all untreated cases tend to progress until lid distortions leading to trichiasis and entropion are produced and the cornea becomes damaged by persistent pannus with or without ulcers at its margin and by catarrhal or phlyctenular ulcers.

Where a trachomatous eye is irritable, or is undergoing a relapse with a congested pannus, too vigorous treatment should not be instituted at first until it is seen how the eye will stand treatment and argyrol 10% to 15% may be used to soothe the eye, but as soon as possible the condition must be attacked by daily scrubbing after cocainization with tooth picks tipped with cotton wool and dipped in *hydrargyri oxycyanidum* (one in 500) and then as the discharge is reduced or the conjunctiva becomes less vascular, daily applications of a copper sulphate pencil. Whatever is done must be done thoroughly, as the consequences of inefficient treatment may be tragic. Expression of follicles may be necessary, but it is doubtful if this comes within the province of a general practitioner.

When the hyperæmia of the conjunctiva and of the pannus is gone and no follicles are seen, treatment at home may be permitted, and for this copper sulphate drops (half a grain to the ounce), or copper citrate ointment (ten grains to the ounce), and tannic acid drops in addition, if a second daily instillation is required, are the most suitable. It is to be impressed on the patient and friends that trachoma is a contagious disease, spread by direct contact or by transference of discharge through the medium of washing water, towels, flies *et cetera*, all the more readily when there is a free discharge in the so-styled acute stages; but, granted suitable and sensible precautions are taken, the danger of infection is not great.

The chronic form, named spring catarrh, is but little amenable to treatment. Astringents may be soothing, but more special treatments, such as by radium and any form of X rays, must be given by specialists in that work, as careless applications are dangerous, and, at the same time, no cure can be guaranteed from their use.

Phlyctenular conjunctivitis is more properly a conjunctivo-keratitis, and it demands a review of the patient's general bodily condition and the hygiene of the living surroundings. Vitamin *A* and life in the open air do much to ameliorate it, but locally mecurial lotions and *unguentum hydrargyri oxidi flavi* 1% are established remedies. The severe forms of ulceration belong to the keratitis group, and are, therefore, not dealt with here.

D. D. PATON, M.A., M.B. (Edinburgh), F.R.A.C.S., D.O. (Oxon),
Honorary Ophthalmic Surgeon, Children's Hospital, Perth.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Anatomy Department, University of Melbourne, on October 4, 1933. The meeting took the form of a series of demonstrations.

Preauricular Fistulæ and Appendages.

PROFESSOR F. WOOD JONES showed specimens and drawings illustrating the development of the external ear. Contrary to the usually accepted account of the origin of the auricle, it appeared that only the tragus was derived from the mandibular arch, the remaining portions of the pinna being hyoid in origin. This was in harmony with the condition of the ear found in cases of agnathia and rendered the explanation of the presence of preauricular sinuses and appendages a simple matter, since the line of the first branchial depression was between the tragus and the rest of the ear. Only the tragus and anterior portion of the external auditory meatus were supplied by the trigeminal nerve, the remainder being cervical in supply.

The Sympathetic Nervous System.

DR. A. E. COATES showed a series of dissections. He demonstrated the segmental innervation of the arterial trunks of the limbs. The fine vascular branches of the peripheral nerves were seen to enter the adventitia of the respective arteries. Microphotographs of the nerves indicated that they contained both myelinated and non-myelinated fibres. The trophic changes and other vascular disturbances seen in lesions of nerves, for example, the *cauda equina*, could be appreciated.

The nerve supply of the urinary bladder was demonstrated. The *nervi erigentes*, the presacral nerve (superior hypogastric nerve), and the hypogastric ganglion were shown.

The abdominal sympathetic trunk and its connexions, including the nerve supply to the lower part of the colon, the cervical sympathetic and the stellate ganglion were displayed.

A preparation of the skull treated with nitric acid and dissected to show the facial nerve and the geniculate ganglion was exhibited. The relations of the cranial nerves, the Gasserian ganglion and the venous sinuses were indicated. The variable relation of the superior petrosal sinus to the sensory root of the Gasserian ganglion was commented upon. The small size of the vagus in the cranial cavity contrasted with its large diameter in the neck and thorax. Dr. Coates said that the latter part of the vagus must be regarded as a different structure from that originating in the medulla.

Attention was drawn to the recent additions to knowledge of the sensory nerve supply of the head and the parts played by the trigeminal, facial and autonomic nerves.

Descent of the Testis.

DR. E. F. HARBISON presented dissections of a series of male fœtuses of various ages to illustrate the descent of the testis. The relative size of the gubernaculum and its attachments was well seen.

Joints of the Lower Limb.

Dr. Harbison's second demonstration was that of the surgical approaches to the joints of the lower extremity. Much interest was taken in the exposure of the knee joint by the oblique patella-splitting incision which was suggested by Devine. For comparison, a preparation showing the patella-splitting exposure advocated by Russell in 1906 was also shown.

Surgical Anatomy of the Abdomen and of the Nasal Sinuses.

DR. W. A. HAILES demonstrated the surgical anatomy of the right side of the upper part of the abdomen from a series of dissections. Dr. Hailes also showed preparations

of the nasal fossæ and paranasal sinuses, and further dissections to show the relations of structures in the floor of the mouth and about the tonsil.

The Thoracic Approach to the Œsophagus.

DR. E. S. J. KING demonstrated dissections showing the relations of the thoracic part of the œsophagus and the surgical approach to this organ. Other dissections were shown to illustrate the advantages of the approach through the left pleura over that through the right. Dissections of the arterial supply of the œsophagus, both in the adult and in the fœtus, were also demonstrated, special attention being directed to those vessels which would cause most difficulty in the operative removal of the organ. Specimens were shown to illustrate complications of carcinoma of the œsophagus, and two specimens of carcinoma removed at operation, one of them from a patient who had lived for sixteen days after operation (death occurred from fulminating pericarditis with effusion).

Mulligan's Stain for Brain Tissue.

DR. EDWARD FORD demonstrated the gross anatomy of the brain from a series of preparations stained by the new method of Mulligan. By this method, which is a variation of the less satisfactory one of Sincke, the brain tissue is differentially stained in a striking manner, which leaves the white matter a clean white, while the grey is stained an intense black. The nuclear masses and fibre tracts are clearly shown, and the method forms a valuable aid to the teaching of the anatomy of the brain.

Fascial Spaces of the Hand.

DR. W. E. A. HUGHES-JONES demonstrated the fascial spaces of the hand in specimens which had been injected with pigmented gelatin. An account of this method was published by Dr. Hughes-Jones in the issue of November 11, 1933.

Osteoplastic Cerebellar Approach.

Dr. Hughes-Jones also demonstrated the osteoplastic cerebellar approach, the chief features of which were:

1. An incision from the spinous process of the sixth cervical vertebra to above the inion, deepening this in the mid-line until all the spinous processes were reached.
2. Exposure and removal by piecemeal nibbling of the arch of the atlas.
3. Gradual removal of the bone behind the *foramen magnum* by piecemeal nibbling.
4. A curved incision in the bone from one mastoid process to the other and extending well above the inion.
5. A vertical mid-line cut in the bone below the transverse incision, commencing with a keyhole saw and completing it with a sharp blow on a chisel.
6. Raising each bone flap by leverage.
7. The puncture of the posterior horn of one lateral ventricle in cases in which the pressure within the posterior fossa was increased.
8. An incision in the dura, over both lobes of the cerebellum, joined in the mid-line after division of the *falx cerebelli* between ligatures.
9. Any decompression required was effected by further removal of the thin bone at the base of the bone flaps. The earlier removal of the arch of the atlas was also important as a measure of decompression.
10. The bone flaps were restored to their original position and firmly secured by wire sutures through small drill holes.

Dr. Hughes-Jones said that the advantages of this approach were excellent exposure of the whole posterior fossa and secure closure. It was, of course, time-consuming.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal North Shore Hospital of Sydney on June 22, 1933. The meeting took the form of a series of clinical demonstrations by the members of the honorary staff.

Tuberculosis of the Knee Joint.

DR. F. GUY GRIFFITHS showed a young woman, aged twenty-eight years, who had been admitted to hospital in 1929, complaining of swelling of the left knee with pain of three years' duration. The patient also complained of cough and loss of weight. The condition was one of tuberculosis of the knee joint and the patient had been treated with tuberculin in doses ranging from 0·00001 to 0·68 cubic centimetre. The case was reported in THE MEDICAL JOURNAL OF AUSTRALIA of October 4, 1930. Dr. Griffiths pointed out that since 1930 the patient had gained 12·6 kilograms (two stone) in weight. There was a little more movement at the knee joint and the patient was able to walk about briskly with a stick.

Case for Diagnosis.

Dr. Griffiths also presented a case for diagnosis. The patient was a woman, aged fifty-five years, who complained of a gnawing hunger pain in the epigastrium. She was cachectic and complained of diarrhœa and vomiting. This case will be reported in full in a subsequent issue.

Progressive Neural Muscular Atrophy.

DR. R. S. ENEVER TODD showed a woman, aged twenty-six years, who gave a history of increasing weakness in the right foot for ten years and in the left foot for six months. Lately she had been unable to walk. The feet were in a position of *equinus* with *pes cavus*; there was loss of power in the peronei muscles of each leg, much greater on the right side. The knee jerks and ankle jerks were absent. All sensations were impaired in the feet, there were no tremors and no clonus, and coordination was good. The Wassermann and Kahn tests gave no reactions.

This patient had two children, one of whom had double *pes cavus* with no other signs who was also exhibited.

The patient's father and two of his sisters, as well as two of the patient's brothers, had "high insteps". The diagnosis was progressive neural muscular atrophy.

Driver's Thigh.

DR. W. WILSON INGRAM showed a man, aged forty-three years, who was suffering from driver's thigh, an occupational right-sided sciatica. The man was a truck driver, conforming to the description of this condition by Dr. John Hoets which appeared in THE MEDICAL JOURNAL OF AUSTRALIA on February 20, 1932. Recovery followed rest, massage and faradism to the wasted muscles. It was pointed out that the exciting cause was faulty construction of the driver's seat in his motor vehicle, while focal sepsis and the predisposing causes of sciatica were the predisposing causes of driver's thigh. The case was later complicated by persistent sciatic scoliosis, an involuntary spasmodic postural phenomenon, which caused residual lumbar pain, and was cured by the application of a light plaster jacket.

Microcephalus.

DR. F. J. BRIDGES showed a child, aged one year, who was suffering from microcephalus. The parents noticed nothing amiss with the child until nine months ago, when it was brought to the casualty department of the hospital. It was then found that the fontanelles had closed. Dr. Bridges pointed out that of late the child had exhibited purposeless movements. Great hyperextensibility of the joints was noted and bilateral *talipes plantaris* was present.

Pediculosis of the Eyebrow.

Dr. Bridges also showed crab lice that had been removed from the eyebrows of a man who had harboured no lice elsewhere.

Cœliac Disease.

DR. C. W. SINCLAIR showed a girl, aged six years, who had been healthy and thriving until five months ago, when she commenced vomiting once every day or every second day. A week later she had a severe cough which lasted a fortnight. Three months later she had an attack of

diarrhœa lasting for three days. From then on her motions assumed the character of those seen in cœliac disease and were passed once or twice a day. The motions were large, pale, greasy, porridgy in appearance, and offensive. Dr. Sinclair pointed out that growth had been irregular, and on the whole the weight had been stationary for the last five months. The composition of the stools was as follows: Total fat 47%, free fatty acid 2·4%, soaps 16%, neutral fat 28·6%. Of the total fat in the stools 39% was split fats and 61% unsplit fats. The abdomen was enormously distended and X ray examination revealed a condition of megacolon. No anæmia was present. The blood sugar was 0·13%, the blood phosphorus was 2·5%, the blood urea was 26·6% and the blood calcium was 10·4%. The skin test for tuberculosis had given no reaction.

Microcytic Anæmia.

DR. ERIC ASPINALL showed a woman, aged thirty-five years, who was suffering from microcytic anæmia. She had six children, the eldest being twelve years and the youngest three and a half. The patient had had no miscarriages, but had had "floodings" for three months. No gynæcological cause for the uterine hæmorrhage could be found. The pelvis appeared to be normal.

Examination of the blood revealed the following:

Erythrocytes, per cubic millimetre ..	4,540,000
Hæmoglobin value	48%
Colour index	0·53
Leucocytes, per cubic millimetre	6,500
Polymorphonuclear cells	63%
Lymphocytes	29%
Monocytes	3%
Eosinophile cells	3%
Basophile cells	2%

The erythrocytes showed variations in size and shape. Many cells were smaller than normal and many appeared deficient in hæmoglobin. No polychromasia was present. No nucleated red cells were seen. There was no abnormality in the white cells. Achlorhydria was present. The patient had been living on a diet deficient in iron.

Pulmonary Tuberculosis.

DR. COTTER HARVEY and DR. A. J. HOOD STOBO showed several patients who were suffering from pulmonary tuberculosis.

Two patients were suffering from senile tuberculosis. The first of these was a male, aged seventy-two years, who had been admitted to hospital after a motor car accident. He complained of a cough with sputum for two months; the cough was worse in the mornings, and the patient had lost weight. On examination of the lungs the percussion note on the left side was impaired. Prolonged expiration was audible at the left apex and moist râles were heard on the left side. The patient had a very troublesome cough with copious sputum and complained of dyspnœa on exertion. X ray examination revealed an old tuberculous lesion with the cardiac shadow drawn over to the left. Tubercle bacilli were present in the sputum.

The second patient with senile tuberculosis was a woman, aged seventy-one years. She had had a cough for many years, but it had been worse during the last ten years. She coughed up about half an ounce of sputum every day. She complained of dyspnœa and had a persistent stabbing pain in the left axilla. On examination bronchial breathing was heard at both apices. The findings on X ray examination suggested an old tuberculous lesion of both lungs with extensive adhesions at both bases. No tubercle bacilli were found in the sputum. The patient's mother died of pneumonia. Two brothers died of pulmonary tuberculosis and one son had the disease.

The third patient shown by Dr. Cotter Harvey and Dr. Stobo was a woman, aged twenty-four years. The patient was first seen in August, 1932, complaining of cough and sputum of three months' duration. Definite signs were present in the right apex. X ray examination at that time revealed tuberculous infiltration of an active type in the right infraclavicular region. Tubercle bacilli were present in the sputum. The patient looked toxic and was of poor physique. Artificial pneumothorax was induced and the patient was in hospital for two months. She made good progress. An X ray examination was made in December, 1932, and the report was to the effect that no evidence of tuberculosis could be seen. Fluid appeared at a later date and the lung became adherent. The patient went to a sanatorium in March, 1933, but soon had a relapse and spent two months in bed. She then returned to the hospital with signs at the site of the old lesion, and X ray examination revealed infiltration greater than that found at the first examination. The patient's mother died of pulmonary tuberculosis and two sisters were suffering from progressing pulmonary tuberculosis.

The next patient was a young woman, aged eighteen years, who had an hæmoptysis in March, 1931. Signs were not definite, but tubercle bacilli were found in the sputum; no examination could be made. The patient was sent to a sanatorium, where she remained for nine months, gaining twenty pounds in weight and losing all her symptoms. In February, 1932, X ray examination failed to reveal the presence of tuberculous changes, but signs of chronic bronchitis were found. She then spent eight months in New Zealand, where she remained well. In November, 1932, X ray examination failed to reveal any signs of tuberculosis.

It was explained that the patient was seen again about four weeks before the meeting. At that time she complained of having had a heavy cold at Easter time with a return of cough. The patient had an hæmoptysis two weeks before being seen. Signs of infiltration were present at the right apex. X ray examination revealed active tuberculous infiltration in the right upper lobe. The patient was being treated by artificial pneumothorax. The family history was that an uncle who lived at the patient's home had pulmonary tuberculosis. A twin sister of the patient had developed pulmonary tuberculosis at almost the same time as the patient; she was being treated by artificial pneumothorax and the disease was quiescent.

Dr. Cotter Harvey and Dr. Stobo then showed two patients who were suffering from bronchiectasis and pulmonary tuberculosis. The first of these was a woman, aged twenty-seven years, who complained of cough with copious sputum that was frequently blood-stained. These symptoms had followed an attack of pneumonia and pleurisy six years previously. On examination there were signs of cavitation at the base of the left lung and doubtful signs at the apices. X ray examination revealed an appearance suggestive of an active tuberculous infection of the right lung. There was a healed tuberculous lesion in the left apex and probably advanced bronchiectasis at the left base.

The other patient was a woman, aged fifty-four years. She gave a history of having had a pulmonary abscess thirty years ago following an anæsthetic for a dental operation. At that time thoracotomy and drainage were used. Since then the patient had had cough with several ounces of sputum every day; the sputum was often offensive. The patient also had recurrent hæmoptysis, sometimes large in amount. On examination there were signs of extensive fibrosis and bronchiectasis at the base of the right lung. A bronchogram revealed small saccular bronchiectases at the right base. There was also a chronic tuberculous lesion at the left apex. Repeated examinations had failed to reveal the presence of tubercle bacilli in the sputum. The patient's mother and one sister had died of pulmonary tuberculosis.

Cœliac Disease and Pulmonary Tuberculosis.

Dr. Stobo showed a boy, aged seven years, who was suffering from cœliac disease and pulmonary tuberculosis. He had had a cough off and on for six years. He had hæmoptysis two and a half years ago. There was no family history of chest trouble. The patient had rickets and bronchopneumonia four years ago; he had bronchopneumonia two years ago and pneumonia three months ago. About five years and one month ago the child's condition was diagnosed as cœliac disease at the Royal Alexandra Hospital for Children. A test of the fæces revealed:

Total fats	54·80%
Split fats	38·49%
(*a*) Soaps	20·60%
(*b*) Fatty acids	17·89%
Unsplit fats	16·31%

A few months later a test of the fæces revealed the following:

Total fats	40·58%
Unsaponified fat	22·36%
Free fatty acid	14·39%
Neutral fats	7·97%

The blood calcium content was 8·3 milligrammes *per centum* and the blood inorganic phosphate content 4·3 milligrammes.

At the time of the meeting the child still had a cough, and Dr. Stobo said that X ray examination of the chest on May 12, 1933, revealed an appearance suggestive of tuberculosis of both lungs, most advanced in the right upper lobe. The radiologist thought that most of the trouble appeared to be acute and fairly recent. Examination of the fæces revealed the following:

Total fats	46·3%
Free fatty acid	2·9%
Carbonized fatty acid ..	0·6%
Neutral fats	43·4%

No tubercle bacilli had been found on examination of the sputum.

Epilepsy in Children.

DR. H. C. MCDOUALL showed two children who were suffering from epilepsy. The first was a boy, aged six years, who suffered from convulsions in infancy and again when he was three years of age. At this time he was unconscious for sixteen hours after eating a green peach. He had further fits seven months and six months before the meeting. The child was well grown and well nourished. He had a rather large rounded head. His speech was thick and he was mentally defective. He was mischievous and he had a slight right internal strabismus. Dr. McDouall said that for six months the child had been taking 0·42 gramme (seven grains) of bromide of potash three times a day and 0·03 gramme (half a grain) of "Luminal" at night. He had had only one fit, lasting twenty minutes, in April, and before this fit he had omitted to take his "Luminal" for three nights.

The second patient was a boy, aged four and a half years. The history was that in November, 1930, the mother stated that the child had been ill for six months. He had had encephalitis six months previously. He could not talk or walk or feed himself, and he dribbled occasionally. Before the illness he had been able to speak and walk. He had epileptic fits, three per night, which occurred at fairly regular times. The optic fundi and cerebro-spinal fluid were normal, and X ray examination of the skull revealed no abnormality. Dr. McDouall said that the child had been taking 0·48 gramme (eight grains) of bromide of potash three times a day and 0·96 gramme (sixteen grains) at ten o'clock at night. He had had no fits for over two years. He talked better, his memory was good, and he was able to feed and dress himself.

Dislocation of the Acromio-Clavicular Joint.

DR. S. H. SCOUGALL showed a man, aged twenty-seven years, who was injured at football on June 17, 1933. While he was lying with the right shoulder touching the ground and the body inclined obliquely, the other players fell on him. On examination there was an obvious and extreme dislocation of the clavicle upwards at the acromio-clavicular joint. The extreme degree of mobility suggested that in addition laceration of the coronoid and trapezoid ligaments had occurred. Dr. Scougall said that it was a general surgical opinion that open operation had to be employed to secure a good functional and cosmetic result. The lesion occurred infrequently and in the past conservative treatment had not been satisfactory. The object of treatment was to overcome the weight of the arm, which drew the acromion downwards and inwards and away from the clavicle, and to counteract the reflex contraction of the trapezius muscle, which drew the clavicle upwards. Trynin, at the Hospital for Joint Diseases, New York, had adapted for this condition the Böhler splint for fracture of the clavicle. Dr. Scougall used this splint in the treatment of the patient, and he said that the advantage to the patient in having free use of the whole extremity while reduction was being maintained was very evident.

Dr. Scougall demonstrated the splint. It consisted of a board thirty centimetres long, fifteen centimetres wide and five centimetres thick. The part fitting into the axilla was smoothed into a Gothic arch in order to fit the conformity of the axilla. Two malleable iron ribbons, 50·0 by 4·0 by 0·4 centimetres, with two short belts of webbing, were fastened to the main part of the splint and were used for passing the splint to the front. A third webbing attached to the splint was fastened over the normal shoulder and was used to raise the injured shoulder. A fourth belt with a buckle was introduced under the lower iron ribbing and was used to press the clavicle down. All parts were well padded and under the webbing used to exert pressure on the clavicle a soft pad was placed. About five cubic centimetres of a 2% solution of "Novocain" were used as a local anæsthetic before the dislocation was reduced. Dr. Scougall said that the shoulder joint, the elbow and fingers were free when the splint was applied and the patient could make immediate use of his hands in any light occupation without displacing the reduction. Atrophy of the muscles was avoided and there was no joint stiffness. If there was pressure in the axilla against the blood vessels and the nerves, the patient only had to raise his arm to release the pressure. Dr. Scougall showed skiagrams to demonstrate the complete reduction of the dislocation.

Thrombo-Angiitis Obliterans.

Dr. Scougall also showed a man, aged fifty-six years, a pensioned fettler, who was suffering from *thrombo-angiitis obliterans*. The patient was quite well until 1920, when, following bruising of the right shin and prolonged exposure in water, two toes became gangrenous. These were amputated, but the condition progressed and fourteen successive amputations had removed both legs to the upper third of the thigh, and the right forearm to near its mid-point. The patient now had pain in the left hand, with threatened gangrene, and had been sent from the country for investigation regarding the advisability of sympathectomy.

Arthrokatadysis of the Hip Joint.

Dr. Scougall showed a patient who was suffering from arthrokatadysis of the hip joint. An account of this case will be published in a subsequent issue.

Apparatus for Continuous Infusion.

DR. E. M. HUMPHERY demonstrated an apparatus for administering solution at an even body temperature. An account of this apparatus will be published in a subsequent issue.

Stricture of the Ureter Causing Fatal Pyelonephritis.

DR. R. J. SILVERTON showed a specimen of a stricture of the right ureter that had given rise to fatal pyelonephritis. The patient was a male, aged forty-nine years, who had undergone suprapubic prostatectomy and left hospital apparently quite well. He returned a couple of weeks later, extremely ill, with pain in the right loin and a swinging temperature. He did not report early enough, and death supervened before any operation, which was considered too risky at the time, could be undertaken.

The specimen showed a definite stricture, about one centimetre long, lying about nine centimetres down the right ureter. Below the stricture the ureter was approximately normal, but above the stricture the duct was thinned out, dilated and congested. The congestion was extreme immediately above the stricture. The kidney showed a little dilatation of the pelvis and calyces, but the outstanding change was an acute focal pyelonephritis, evidenced by suppurative foci in the parenchyma. On the surface of the organ these areas are also seen. The specimen shown was removed at autopsy.

Acute Focal Pyelonephritis.

Dr. Silverton also showed a kidney which was the site of acute focal pyelonephritis and which had been removed from a female, aged thirty-eight years. The patient gave a two months' history of pain in the right loin, swinging temperature, vomiting, rigors occasionally, and some pain at the end of micturition. Cystoscopy showed no efflux on the right side, and the intravenous urogram revealed well marked dilatation of the calycine system on the right side. The patient's condition was ameliorated by draining the right kidney for several days by retained ureteric catheter, through which urine thick with pus was evacuated. Nephrectomy was performed a few days later.

The specimen did not show so much dilatation as the pyelogram indicated, but the acute pyelonephritic process was well seen on the surface of the kidney, where islands of suppurative areas were distinct. There was no obstruction at the pelvic outlet. There was a chronic pyelitis and pyelonephritis, with invasion of the renal hilum by fat. On these chronic processes the acute infection supervened.

Early Renal Tuberculosis.

Dr. Silverton also showed a kidney that had been removed from a female, aged twenty-four, who suffered chiefly from a painful contracted bladder. A valuable part of the diagnosis was the intravenous urogram, which showed a dilated ureter and stunted calyces on the right or affected side, while the upper part of the urinary tract on the left side appeared normal. Cystoscopy was quite unsatisfactory, even under a spinal analgesic, so nephrectomy had to be undertaken on the urogram findings.

At operation the kidney seemed to be fairly normal externally, but nephrectomy was proceeded with. Section of the specimen showed very early tuberculosis affecting only the apices of a few of the pyramids.

Pneumopyelography.

Dr. Silverton showed three radiograms illustrating the value of pneumopyelography in localizing calculi in the kidney, and also in actually determining that they are calculi.

1. A large, irregularly ovoid calculus was very plainly seen to be lying in a somewhat dilated renal pelvis.

2. The plain radiogram showed two small, irregularly star-shaped shadows in the renal area. Pneumopyelography definitely showed them to be caused by calculi, and localized the calculi to the middle and lower sets of calyces respectively. After air was injected and allowed to escape, Dr. Silverton said that it was his practice to fill the pelvis and caylces with 5% sodium iodide solution and make a chemo-pyelogram. In this case the calculi were overlarded by the iodide and were therefore not definitely localizable.

3. A fairly large, rather Y-shaped calculus, with thick blunted limbs, was localized to two branching limbs of a dilated lower set of calyces. In the pneumopyelogram the position of the calculus was apparent, while in the iodide pyelogram the calculus appeared moderately clearly as an area of relatively less density.

Renal Ptosis Illustrated by Pyelography.

Dr. Silverton showed two pyelograms, one cystoscopic and the other intravenous, showing kinking of the upper end of one ureter due to renal ptosis. Early dilatory effect on the kidney was illustrated by slight expansion of the pelvis in each case, broadening of the major calyces and clubbing of the minor calyces.

Crossed Renal Dystopia with Fusion.

Dr. Colin Edwards showed a woman who was suffering from crossed renal dystopia with fusion and in whom heminephrectomy had been performed. It is hoped that this case will be reported in a subsequent issue.

A meeting of the Section of Surgery of the South Australian Branch of the British Medical Association was held at the Adelaide Hospital on July 12, 1933, Dr. A. M. Cudmore, the President, in the chair. The meeting was devoted to a series of clinical and pathological demonstrations.

Jacksonian Epilepsy.

Sir Henry Newland showed an Australian native from a tribe in Central Australia who, three years previously, in the course of a fight with his brother, had been struck on the left side of the head with a boomerang. Since then he had occasional Jacksonian attacks and persistent paresis of the right upper extremity.

On examination there was a scar over the situation of the arm centre on the left side of the head, with a very definite linear depression in the underlying parietal bone. A skiagram showed a depression encroaching on the cranial cavity in this situation. The movements of the right hand, forearm and arm were all definitely weaker than on the left side.

While the patient was in the Adelaide Hospital definite Jacksonian attacks without loss of consciousness occurred.

The operation consisted in turning down an osteoplastic flap. When freed from the dura, the bone was found to be depressed for eight millimetres (one-third of an inch) at the site of the injury. This bone was removed with bone forceps. The dura in the same situation was adherent to the pia-arachnoid over an area of about six millimetres (one-quarter of an inch). A circular incision was made and the adherent dura dissected away. There was a little bleeding, which was checked by muscle pledgets from the temporal muscle. A piece of pericranium was placed over the area from which the dura had been excised. The bone flap was replaced and the scalp sutured in layers. Some Jacksonian attacks had occurred since the operation, and it was too early to say whether a cure would result.

Carcinoma of the Larynx.

Sir Henry Newland's second patient was a man, aged sixty years, who had been admitted to the Adelaide Hospital suffering from a malignant growth in the region of the right ventricle of the larynx and extensive secondary deposits in the right cervical lymph glands. The latter had been removed by an extensive dissection. Later on a lateral pharyngotomy was performed and radium needles were inserted into the growth. The lateral pharyngotomy wound had been kept open so that the effect of radium on the growth might be observed.

Cinematograph Films in Colour.

Sir Henry Newland also showed a series of Kodacolor cinema films. The following subjects were illustrated: an operation for supposed depressed fracture of the skull, a case of chronic gangrene of the abdominal wall, removal of a tumour of the auditory nerve, and removal of an appendix.

Sir Henry Newland stated that his interest in the subject had been stimulated by Professor Kelly, of Liverpool, during his recent visit to England. With the valuable assistance of Kodak Proprietary, Limited, the technique had been developed. He thought that the films showed that surgery offered a field for the useful employment of colour cinematography.[1]

Squamous-celled Carcinoma Developing in Chronic Ulcer of the Leg.

Dr. Leonard Lindon showed a patient, aged sixty-eight years, who had suffered from chronic recurring ulceration of both shins for the last thirty-five years. His occupation

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[1] Kodak (Australasia) Proprietary, Limited, have kindly forwarded the following statement. [Editor.]

The use of substandard (16 millimetre) film for recording medical and surgical subjects has steadily advanced step by step with the improvement of apparatus and sensitive material. With the introduction of Eastman super-sensitive panchromatic film some time ago, the making of Ciné-Kodak records of operative surgery has been greatly simplified. The new emulsion has made it possible to obtain excellent photographic results with very little more than the normal artificial lighting of the operating table.

A further great step in advance has now been made, for moving picture records in natural colours are easily within reach of any surgeon who has an F/1·9 Ciné-Kodak, the new Kodacolor film and Photoflood lamps for illumination.

for many years had included that of shoeing horses, and the frequent knocks so received had rapidly opened up the healed ulcers. For the past fifteen years he had been an inmate of the Home for Incurables. In January of this year he had been referred to the surgical ward from the department of dermatology, owing to the presence of a large area of apparent hyperplasia of the marginal tissues of the indolent ulcer on the right leg. This proved to be a squamous-celled carcinoma supervening on a chronic ulcer. The leg was amputated just below the knee joint, leaving sufficient of the tibia to give him a flexible stump, one which at least would be capable of accommodating a peg leg. His convalescence was uneventful, and he was discharged to the Home for Incurables.

In the month of July, 1933, he was again admitted to the surgical ward, with the request for amputation of his remaining leg. This leg he had not put to the ground for twelve years, and he was now suffering a good deal of pain in the leg and had become wearied of the repeated dressings and had decided to ask for removal of the limb. It was noticed that at one point in the margin of the very extensive ulcer (which almost entirely encircled the leg above the ankle and extended for about fifteen centimetres up the leg) there was a suspicious hyperplasia of tissues; on section this again proved to be a squamous-celled carcinoma. The leg was amputated after a manner similar to that which was pursued in the case of his right leg.

Dr. Lindon said that it was unusual for chronic ulcer of the leg to become carcinomatous; it must be extremely unusual to find this condition occurring in each leg of the same patient. On neither side had enlarged lymph glands been discovered, and so far no treatment had been directed to the glands in either groin.

Carbuncle of the Nose.

DR. IAN HAMILTON showed a patient who, as a robust, healthy man aged twenty-five years, had consulted him on June 25, 1933, complaining that two days previously he had noticed a pimple just inside his nose. This had been treated by a process of squeezing. The following day the nose was very sore, but he felt well and played a good game of football that afternoon, it being Saturday. The nose was exposed to a bitter wind during the game and was worse afterwards. Twenty-four hours later its appearance alarmed him and he decided to see a doctor.

On examination the patient's temperature was 37·2° C. (99° F.). On the edge of his right nostril there was a large pimple with a necrotic centre. The whole nose was evenly swollen, bright red and shiny, and tender to touch. He was sent home after having the pimple cleaned out and cauterized with pure carbolic acid, and was given some ammoniated mercury ointment to rub into the nose. Next day he felt ill, and when seen his temperature was 37·7° C. (99·9° F.) and his pulse rate 92. The whole nose was very swollen, and extending up from its tip near the origin of the original pimple there was a row of pustules coming through from the subcutaneous tissue. They could be seen shining through. The nose resembled a strawberry of large dimensions with white spots. He was sent to hospital and hourly fomentations were applied. Next day the whole nose was covered with pustular points. At this stage, after desensitization, 90 cubic centimetres of antistaphylococcal serum were given, it having been determined that a *Staphylococcus aureus* was the cause of the infection. The dose was given subcutaneously. A day later both eyes began to close, the swelling extending out to the supraorbital and infraorbital areas. His temperature was still elevated in the neighbourhood of 37·8° C. (100° F.) and his pulse rate was about 90 per minute. The nose looked like a sponge filled with pus, with a number of points on the surface, but when opened they discharged only a little pus and fresh ones appeared to replace them.

That evening, in lieu of tying the angular veins, as it appeared that the infection was spreading further afield and might very easily lead to sinus thrombosis, a ring of soft tissue around the base of the nose was coagulated with the diathermy button. This was intended to coagulate the angular veins on both sides and all soft tissue by which the infection and necrosis might spread upwards. This was the fifth day of the illness. Next day both eyes were closed, the operation having increased the swelling. Pustules appeared right up to the line of coagulation, but apparently the staphylococci did not go beyond that line, because within a few days all swelling on the healthy side of the line subsided and the eyes opened again. But about three days later a large swelling developed in the left paratoid region and the whole of the left side of the face took part in it and the left eye then closed again. The main part of the swelling near the parotid was very hard; and as it was thought to soften in the centre after a couple of days an incision was made into it, but no pus could be discovered. This was done on the sixth day after the diathermy was applied. Serum drained out. At the same time, as the nose still looked like a sponge filled with pus, an incision was made into each side of its dorsum, but these only confirmed the appearance, and in actual fact it was of the nature of a pus-filled sponge and it was impossible to drain it.

Within another two days the swelling of the left side of the face began to subside, and then two days later again a hard swelling came up and appeared to concentrate in the left infraorbital region. Dr. Hamilton said that although he was tempted to make an incision, the experience of opening the swelling in the parotid had taught him to wait, and within a couple of days of its appearance this swelling began to subside. The left eye had once again opened and shut and now opened again gradually. The condition of the nose gradually resolved; the pustules appeared to absorb without discharging much. After about ten days the coagulated ring sloughed away, healthy granulations appeared, the nose, which was completely blocked for a week or more, gradually cleared away, and except for the manifestations of serum disease, which appeared seven days after the administration and continued to appear for more than a week, the patient made a steady recovery.

The nose was completely healed on July 24 and the patient was fit to resume work a week later.

Dr. Hamilton said that as such acute spreading staphylococcal infections of the face were almost invariably fatal, it was thought that bold measures were necessary in the attempt to save the life, and it was considered that in all probability the operation of interposing a ring of coagulated tissue around the base of the nose prevented any spread of infection towards the sinuses or the meninges, while the patient's resistance, aided by the antinecrotoxic and antileucocidic properties of the serum, was mobilized to deal with the infection. The serum employed was the antistaphylococcal serum prepared by the Commonwealth Serum Laboratories.

The case was demonstrated to the meeting by means of a natural colour moving picture taken by Kodak, Limited, and the great importance of such staphylococcal infections was stressed. In view of the recent work done upon the toxin-producing qualities of staphylococci and the production of antiserum, it seemed that all such cases should be reported in order that the clinical value of serum might be estimated.

Kümmel's Disease of the Spine Involving Three Vertebræ.

Dr. Hamilton's second patient, a woman of fifty-six years, had been involved in a motor accident six years previously. She was sitting in the back seat of the motor car when it ran into a stationary object and she was thrown forward and badly shaken. After three days in bed she was allowed up, and apparently her doctor did not detect any abnormality of the spine at the time. She was told that she would be all right, and no X ray examination was made. From that time she always suffered from "rheumatism" in the back, and during the last three years it had become much more constant and aggravating. The pain was worse on exercise and kept her awake at nights. Her doctor had eventually told her that he could not do any more for her rheumatism, and sent her to the Adelaide Hospital.

On examination she was an elderly woman. There was a marked general kyphosis of the dorsal spine, with a projection of the eleventh spinal process. There were no neurological signs.

A diagnosis of Kümmel's disease was made, and an X ray examination was requested. The radiologist reported on the first skiagram that there was compression of the eleventh dorsal vertebra, with some chronic spondylitis. The upper portion of the picture was somewhat obscured, but on close examination it could be seen that the eighth and ninth thoracic vertebræ were also affected, and on taking some more pictures higher up about three months later the report was:

> There is compression of the bodies of T8 and 9 similar to that of the body of T11. There is some advance in the osteoarthritic changes. There is calcification of the wall of the aorta. The deformities of the bodies of the vertebræ are out of proportion to the spondylitic changes observed. They are, however, apparently different manifestations of the same ætiological factor and represent delayed compression fractures of the bodies of several vertebræ. Such a phenomenon would be consistent with marked degenerative changes in the blood vessels. The forearm bones show unusual bowing of the shaft of the radius without alteration in bone texture. The appearances may indicate a very rare condition, namely, Kümmel's disease in the bodies of several vertebræ.

Dr. Hamilton said that the case was one of considerable interest. The patient had been fitted with a spinal support and had been treated with radiant heat and massage to the spine. This had relieved her pain somewhat and the disease had not progressed any further. The blood calcium and phosphorus were normal.

Crush Fracture of Vertebra.

DR. L. O. BETTS showed a girl, aged eleven, with a crush fracture of the fourth lumbar vertebra, sustained by a fall from a horizontal bar.

The diagnosis had not been made until seven weeks after the injury, when she was placed in a plaster jacket in hyperextension to relieve pressure upon the fractured body. No reduction of the fracture had been obtained.

The case was shown to illustrate the treatment of crush fractures of the spine by the method introduced by Mr. Watson Jones.

Suppurative Pyelo-Nephritis Associated with Impaction of a Stone at the Junction of a Double Ureter.

DR. JOHN CLOSE showed a private patient, a woman, aged thirty-four years. When first seen she was six months pregnant. She had been ill for two or three days, with high temperature, pain in the right loin, and increased frequency of micturition.

A previous pregnancy had been associated with pyelitis, which had subsided under medical treatment. The urine was only mildly purulent. The pulse was rapid, the tongue was dry and furred, and extreme toxæmia was present. Catheterization of the right ureter was impossible beyond seventeen centimetres. The operation of uretero-nephrectomy was performed. There were abscesses in the cortex of the kidney, mostly confined to its upper half. The patient recovered.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the Tasmanian Branch of the British Medical Association:

Kingsmill, Caroline Mary, M.R.C.S., L.R.C.P., M.B., B.S., 1927 (London), St. Helens.

Gollon, Lachlan, M.R.C.S. (England), L.R.C.P. (London), 1900, Ulverstone.

The undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Hayward, John Isaac, M.B., B.S., 1933 (Univ. Melbourne), Melbourne Hospital, Melbourne, C.1.

Schlink, Franziska, M.B., B.S., 1932 (Univ. Melbourne), Melbourne Hospital, Melbourne, C.1.

Williams, Constance Isabel Arundel, B.A., M.B., B.S., 1932 (Univ. Melbourne), Melbourne Hospital, Melbourne, C.1.

Obituary.

ROBERT JOHN FULLERTON.

DR. ROBERT JOHN FULLERTON, who, as recorded recently in this journal, died from injuries sustained in an accident, was born at Carlton, Victoria, in 1868. His early education was undertaken at King's College, Carlton, where he was *dux* in 1886. He studied first of all at Melbourne and later at Edinburgh University, and in 1895 became a licentiate of the Royal College of Physicians and of the Royal College of Surgeons of Edinburgh and of the Faculty of Physicians and Surgeons of Glasgow. On his return to Australia he became resident medical officer at the Kyneton Hospital. He then started practice at Bendigo, where he remained till 1911. After spending a few years at Trafalgar he went to South Melbourne and stayed there till 1931. Early in 1933 he took up practice at Reservoir. During his student days Robert John Fullerton was a keen sportsman; he won his "blue" for football at the University of Melbourne. He was a prominent freemason.

WILLIAM ROBERT BOYD.

WE regret to announce the death of Dr. William Robert Boyd, which occurred on November 28, 1933, at Richmond, Victoria.

ERNEST ADOLPHUS WOODWARD.

WE regret to announce the death of Dr. Ernest Adolphus Woodward, which occurred at Roseville, New South Wales, on November 30, 1933.

Correspondence.

"WHEN HALF GODS GO."

SIR: The late William Archer, mediocre playwright and excellent critic, used to say that a good drama should have these three qualities: (i) It should be true to the visible and audible surfaces of life; that is, the events should be reasonable and probable. (ii) It should beget in the audience high suspense and vivid realization; the mechanism of the theatre must be fully exploited to gain this end. (iii) The hearer or reader must feel that he has undergone an experience and not just attended a pastime or an entertainment.

Tragedy deals with the downfall of a noble character through some human failing that is avoidable, not inevitable. "When Half Gods Go" therefore contains the essence of tragedy. But, judged on Archer's criteria, it cannot be said to be good drama. The scene in the doctor's waiting room is certainly not true to the visible and audible surfaces of life, and the operating scene is obviously introduced merely to satisfy the public taste for that sort of thing. The final scene is hopelessly ludicrous, although it was plainly meant to be overwhelming; spiritualism, rebuked in the first scene, apparently triumphs in the last one, unless I, and many others, missed some subtle point. I cannot agree with "M.D.'s" opinion that the structure of this drama is generally successful.

Yours, etc.,

"AMATEUR CRITIC."

Sydney,
November 24, 1933.

Books Received.

HANDBOOK OF PHYSIOLOGY, by the late W. D. Halliburton, M.D., LL.D., F.R.C.P., F.R.S., and R. J. S. McDowall, M.B., D.Sc., F.R.C.P.; Twenty-Third Edition; 1933. London: John Murray. Medium 8vo., pp. 982, with numerous illustrations in the text, many of which are coloured, and four coloured plates. Price: 18s. net.

POCKET MONOGRAPHS ON PRACTICAL MEDICINE: PULMONARY TUBERCULOSIS IN GENERAL PRACTICE, by A. Morland, M.D., M.R.C.P.; 1933. London: John Bale, Sons and Danielsson, Limited. Foolscap 8vo., pp. 119. Price: 2s. 6d. net.

WILHELM CONRAD RÖNTGEN AND THE EARLY HISTORY OF THE ROENTGEN RAYS, by O. Glasser, with a chapter by M. Boveri; 1933. London: John Bale, Sons and Danielsson, Limited. Crown 4to., pp. 494, with illustrations. Price: 32s. 6d. net.

TUBERCULOSIS: ITS CURE AND PREVENTION, by G. Tippett, M.B.; 1933. London: Methuen and Company, Limited. Crown 8vo., pp. 253. Price: 7s. 6d. net.

FRONTIERS OF MEDICINE, by M. Fishbein, M.D.; 1933. Baltimore: The Williams and Wilkins Company; London: Baillière, Tindall and Cox. Crown 8vo., pp. 217. Price: 5s. net.

VACCINE THERAPY IN ACUTE AND CHRONIC RESPIRATORY INFECTIONS, by H. T. Gillett, M.D., with a foreword by W. H. Wynn, M.D., F.R.C.P.; 1933. London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 116, with nine charts. Price: 5s. net.

THE PHYSICIAN AS A MAN OF LETTERS, SCIENCE AND ACTION, by T. K. Monro, M.A., M.D.; 1933. Glasgow: Jackson, Wylie and Company. Demy 8vo., pp. 220. Price: 10s. 6d. net.

Diary for the Month.

DEC. 12.—New South Wales Branch, B.M.A.: Ethics Committee.
DEC. 14.—Victorian Branch, B.M.A.: Council.
DEC. 14.—New South Wales Branch, B.M.A.: Branch.
DEC. 15.—Queensland Branch, B.M.A.: Council.
DEC. 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments.

Dr. J. M. Sanderson has been appointed Medical Officer of Health for the Esperance Road Board, Western Australia.

* * *

The undermentioned appointments have been made to the staff of the Royal Alexandra Hospital for Children, Sydney, New South Wales: Dr. E. H. M. Stephen (B.M.A.), Clinical Lecturer in the Medical Diseases of Children; Dr. P. L. Hipsley (B.M.A.), Clinical Lecturer in the Surgical Diseases of Children; Dr. J. Shedden David, Honorary Surgeon; Dr. T. Y. Nelson (B.M.A.), Honorary Assistant Surgeon; Dr. J. Steigrad (B.M.A.), Honorary Relieving Assistant Surgeon. The following reappointments have been made for a further four years: Dr. F. C. Rogers (B.M.A.), Honorary Surgeon; Dr. C. H. Wesley (B.M.A.), Honorary Assistant Surgeon; Dr. A. J. P. Chapman (B.M.A.), Honorary Assistant Dermatologist; Dr. D. G. R. Vickery (B.M.A.), Honorary Relieving Assistant Surgeon.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi and xvii

PARRAMATTA DISTRICT HOSPITAL, PARRAMATTA, NEW SOUTH WALES: Junior Resident Medical Officer.

PUBLIC SERVICE COMMISSIONER, ADELAIDE, SOUTH AUSTRALIA: Medical Officers.

ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officers.

SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers.

THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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